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EXPERIMENTAL STUDIES CONCERNING THE NATURE OF HYPERTENSION*

Their Bearing on Surgical Treatment

STANLEY J. G. NOWAK, M.D.,† AND IRVING J. WALKER, M.D.‡

BOSTON

EXPERIMENTAL investigation into the cause of hypertension has followed two lines, one dealing with mechanisms producing increased vasomotor activity, the other with the kidney as a possible source of a hypertensive substance.

HYPERTENSION DUE TO INCREASED VASOMOTOR ACTIVITY

Carotid-Sinus Denervation and Aortic-Depressor Nerve Section. Attempts at producing increased vasomotor tone analogous to essential hypertension have met with success through carotid-sinus denervation and section of the aortic-depressor nerve. First described by Hering's pupils, Koch and Mies,[1] in the rabbit in 1929 and by Heymans[2] in the dog in 1931, this method has been corroborated by Kremer, Wright and Scarff[3] and by one of us (S. J. G. N.[4]). The principle underlying this experimental means for producing hypertension depends upon the interruption of afferent nerve impulses which normally buffer or depress the activity of the circulatory centers in the medulla. Removal of these impulses permits the vasoconstrictor, cardio-accelerator and adrenine-secretory centers to exert their maximal effects, with a resulting elevation of blood pressure in dogs from the average normal level of 130 mm. of mercury to as high as 250 mm. The majority of these animals show oscillations in blood-pressure values similar to those frequently seen in clinical essential hypertension. It has been demonstrated that the hypertension so produced may persist for three years or more.[4]

The obvious advantage of using this group of animals for study is that they represent a hypertensive state due chiefly to vasoconstriction and to a lesser extent to increased secretion of adrenine and cardiac acceleration, factors which are undoubtedly responsible for clinical essential hypertension. It should not be inferred, however, that the clinical condition is due to decreased activity or suppression of the carotid-sinus and aortic-depressor nerve mechanisms. The similarity lies in the end result rather than in the cause.

Chronic Cerebral Anemia. The conception that acute cerebral anemia and asphyxia produce a temporary increase in blood pressure through increased vasomotor activity has been frequently demonstrated since the early days of physiology.[5, 6] By progressive ligation of the various cerebral arteries one of us (S. J. G. N.) demonstrated for the first time that chronic cerebral anemia may result in chronic hypertension (Fig. 1). The difficulties attendant upon this experimental production of chronic hypertension lay in the extraordinary ability of the dog to re-establish practically normal cerebral circulation through well-developed anastomoses, in spite of ligation of both external and internal carotids, both vertebral arteries and the anterior spinal artery.

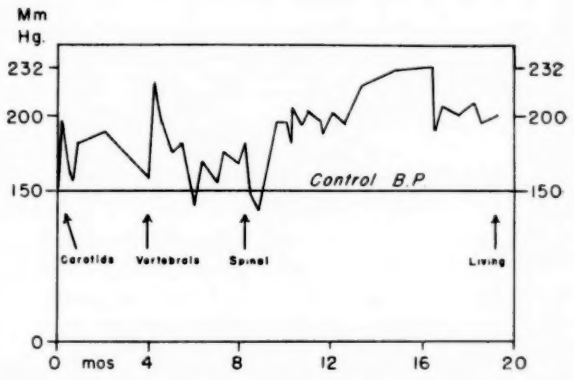

Figure 1. *Hypertension Produced by Ligation of Cerebral Arteries (Dog 20).*

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†Associate in surgery, Harvard Medical School; associate surgeon, Boston City Hospital.

‡Clinical professor of surgery, Harvard Medical School; surgeon-in-chief, Fifth Surgical (Harvard) Service, Boston City Hospital.

Increased Intracranial Pressure. Also in line with this work are the investigations on the effect of increased intracranial pressure on arterial tension. According to Cushing,[7] cerebral compression produces hypertension by virtue of cerebral anemia. His observations in acute experiments and clinical studies have been confirmed by Dixon and Heller,[8] who have produced chronic hypertension by increasing intracranial pressure by means of injecting an inert substance, kaolin, into the fourth ventricle of rabbits.

The mechanism by which cerebral anemia stimulates the vasomotor center has been recently shown by Raab[9] to be due to acidosis of this center; its perfusion by acids, such as lactic acid, has resulted in acute hypertension. On the other hand, perfusion with alkaline solutions brought about a lowering of blood pressure.

Experimental Arteriosclerosis. Handovsky and Goormaghtigh[10] of Heymans's laboratory have observed hypertension following the production of marked arteriosclerosis in dogs by means of the feeding of vitamin D combined with thyroidectomy. It is probable that this hypertension is due to increased activity of the vasomotor center in response to augmented peripheral resistance.

RENAL HYPERTENSION

Since the postulation made by Johnson[11] in 1868, based on clinical observations, that the diseased kidney liberates a vasoconstrictor substance, many investigators have attempted to isolate this hypertensive factor. The first of these investigations was carried out in 1898 by Tigerstedt and Bergman,[12] who extracted a pressor substance from normal rabbits' kidneys which they called "rennin." Similar extracts have been described by Pearce,[13] Bingel and Strauss[14] and others. Recently Landis and his co-workers[15] have isolated a saline kidney extract in rabbits which, on injection into normal unanesthetized rabbits, elevated the blood pressure without diminishing the skin temperature. This absence of a change in skin temperature, indicating as it does undiminished peripheral flow, is characteristic of most forms of human hypertension. It is thus distinctly different from the group of hypertensive substances such as adrenalin, tyramine, pituitrin and so forth which produce hypertension with disproportionate increase of peripheral resistance and therefore of blood flow.

Another approach to the kidney as an etiologic factor in hypertension has been the attempt to establish high blood pressure in animals by inducing changes in the kidney. These changes have been accomplished by the following methods:

Resection of large portions of both kidneys.[16]
Ligation of branches of the renal artery.[17, 18]
Partial occlusion of the renal artery or its branches.[19]
Partial occlusion of the renal vein.[20]
Partial obstruction of the ureters.[21]

The outstanding experimental work in this field is that of Goldblatt and his co-workers,[19] who have produced a marked degree of hypertension in dogs by partially occluding the renal arteries with adjustable metal clamps. These investigators have produced striking elevations of blood pressure in their dogs, as high as 250 to 300 mm. of mercury, for as long as fifteen months.

The nature of the hypertension caused by renal ischemia is revealed by the investigations of Houssay[22] and Blalock and Levy,[23] who successfully transplanted one ischemic kidney into the neck of a dog by anastomosing the renal with the carotid-jugular vessels. The development of hypertension after the transplantation and its abolition by removal of the transplanted kidney point definitely to the kidney tissue as a primary source of the pressor substance.

There is contradictory evidence as to the ability of extracts of the ischemic kidneys to produce hypertension in animals. Harrison, Blalock and Mason[24] and Prinzmetal and Friedman[25] were able to demonstrate hypertensive responses to the injection of these extracts. Page,[26] on the other hand, obtained entirely negative results.

That the endocrine glands are definitely concerned in hypertension of renal ischemic origin is borne out by the findings of Page and Sweet,[27] who showed that this type of hypertension is reduced to normal by removal of the pituitary gland. Goldblatt[28] and Blalock and Levy[23] showed that removal of both adrenal glands also causes a reduction of renoischemic high blood pressure to normal.

These important demonstrations suggest the possibility that hypertension of renal origin is due to a hormone which has its origin in either of these glands and which is activated by a substance liberated by a diseased or ischemic kidney.

EFFECT OF VARIOUS SYMPATHETIC PROCEDURES IN EXPERIMENTAL "ESSENTIAL" HYPERTENSION PRODUCED BY CAROTID-SINUS DENERVATION AND SECTION OF THE AORTIC-DEPRESSOR NERVE

In 1923 Daniélopolu[29] and Brüning[30] made independent suggestions of the possible value of splanchnic-nerve resection in arterial hypertension. Unilateral resection for this condition was first performed in 1930 by Pieri.[31] In 1934 Craig and Brown[32] resorted to bilateral splanchnic resection. This field has been extended by Fralick

and Peet,[33] Smithwick,[34] Page and Heuer[35] and others.

In 1930 Adson and Craig[36] and in 1937 Page and Heuer[37] resorted to extensive ventral rhizotomy in order to obtain a larger area of vasodilatation than that afforded by splanchnic section.

Contemporaneously with the early work in this clinical field one of us (S.J.G.N.[38]) approached the rationale of partial sympathetic exclusion in dogs with denervated carotid-sinuses and sectioned aortic-depressor nerves. It was demonstrated in 1934 that bilateral splanchnic-nerve section combined with bilateral abdominal ganglionectomy failed to reduce the arterial hypertension produced by this means. In the same year Bacq, Brouha and Heymans[39] showed that total sympathectomy in dogs prevented the development of acute hypertension of carotid-sinus, aortic-depressor-nerve origin.

These investigations have been extended and permit further analysis. The results may be best illustrated in Dog 11 of our series (Fig. 2). This dog showed a persistent elevation of blood pressure varying between 180 and 226 mm. of mercury for seventeen months after carotid-sinus and aortic-depressor-nerve exclusion. Operative procedures on the sympathetic system showed the following effects on blood pressure.

Bilateral splanchnic section caused a drop in pressure from 205 to 170 mm. of mercury which lasted only two weeks, when the pressure regained its former level; it subsequently rose to an even higher point.

Extirpation of the right adrenal gland six months later produced a marked but also temporary fall in blood pressure to 60 mm. of mercury, with return to its former level of 190 mm. two months later.

Bilateral abdominal sympathectomy (first lumbar to first sacral) also caused a temporary fall in pressure to 155 mm., with return to its former high level of 205 mm. after two months.

Right thoracic ganglionectomy (stellate ganglion to the eighth dorsal ganglion) two and a half months after the latter procedure caused only a slight fall in pressure (205 to 185 mm.), with a subsequent rise to 225 mm.

This dog died spontaneously five and a half months later, after three years of hypertension, with a final pressure of 205 mm.

The ineffectiveness of bilateral splanchnic-nerve section in reducing this type of hypertension was demonstrated in two other dogs. One of these (Dog 14) with an oscillating type of hypertension showed a more lasting fall in blood pressure from 200 to 156 mm. for five months, with subsequent return to levels as high as 245 mm. (Fig. 3). In the third dog (Dog 7), bilateral splanchnic-nerve section combined with removal of the right abdominal sympathetic chain caused the blood pressure to drop temporarily from 220 to 158 mm. Two weeks later it returned to 190 mm. Nine months later it reached 235 mm. (Fig. 4).

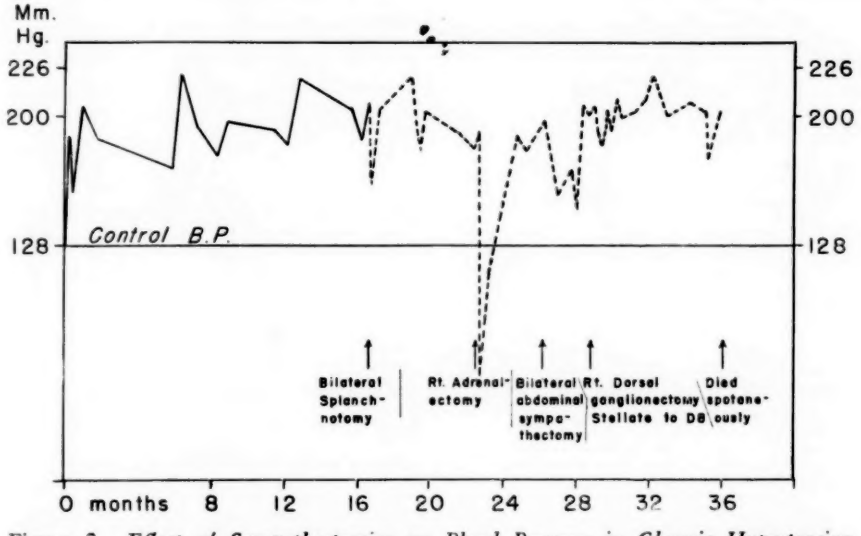

Figure 2. *Effect of Sympathectomies on Blood Pressure in Chronic Hypertension Produced by Carotid-Sinus and Aortic-Depressor Denervation (Dog 11).*

Heymans[40] showed that total sympathectomy carried out in three stages in a dog with previously denervated carotid-sinus and aortic-depressor regions and with resulting hypertension reduced the blood pressure to normal. He observed that the blood pressure still remained at its hypertensive level after three-fourths sympathectomy. This finding is in agreement with our observations on the first dog discussed, which showed a tension of 205 mm. after two-thirds sympathectomy.

By reversing the procedure followed by Heymans we have demonstrated that preliminary total

sympathectomy fails to neutralize experimental chronic hypertension of carotid-sinus and aortic-depressor-nerve origin. Dog 30 was totally sympathectomized in two stages, the entire thoraco-abdominal chain being removed intact in each

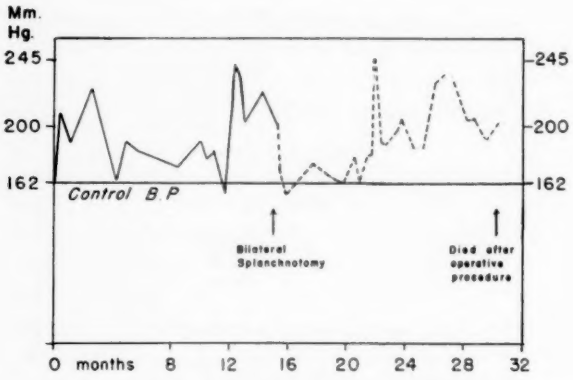

Figure 3. *Effect of Sympathectomies on Blood Pressure in Chronic Hypertension Produced by Carotid-Sinus and Aortic-Depressor Denervation (Dog 14).*

stage, thus eliminating any known sympathetic activity. The control blood pressure was 116 mm. One and a half months after total sympathectomy the carotid-sinuses were denervated and the aortic-depressor nerves sectioned. This procedure was followed by a definite and sustained elevation of blood pressure to 170 mm. The dog died spontaneously two and a half months after denervation, with a blood pressure of 160 mm. (Fig. 5).

Thus bilateral splanchnotomy fails to reduce, except temporarily, a type of experimental hypertension which is preponderantly, if not purely, vasomotor in origin. In fact, two-thirds to three-fourths sympathectomy fails to affect this hypertension. Finally, the mechanism underlying this hypertension is still able to exert a moderate but definite hypertensive effect in spite of total sympathectomy. Whether, in the latter case, this increment in pressure is due to the liberation of some intrinsic factor or to the development of vasoconstriction by some unknown mechanism cannot be answered as yet. It is definitely not due to any organic change such as arteriosclerosis.

From these data one is obliged to draw the conclusion that there is little if any rationale for splanchnotomy and rhizotomy in clinical essential hypertension.

EFFECT OF VARIOUS SYMPATHETIC PROCEDURES ON EXPERIMENTAL HYPERTENSION PRODUCED BY RENAL ISCHEMIA

The effect of partial and total sympathectomy on the nephroischemic hypertension is, as one would expect, completely negative. In this type of hypertension the underlying mechanism is a vasoconstrictor substance liberated by an ischemic kidney which acts on the peripheral vascular musculature. In the presence of this intrinsic stimulus the blood vessels obviously cannot respond to a neurogenic vasodilator mechanism.

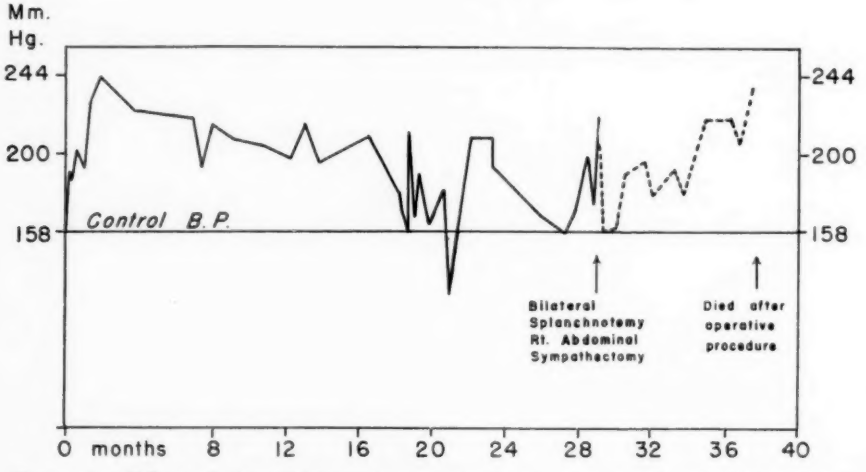

Figure 4. *Effect of Sympathectomies on Blood Pressure in Chronic Hypertension Produced by Carotid-Sinus and Aortic-Depressor Denervation (Dog 7).*

These views are based on the demonstration by Page[41] that denervation of the kidneys does not alter the course of nephroischemic hypertension. Goldblatt[42] showed further that bilateral splanchnotomy is likewise ineffective. In fact, total sympathectomy has failed to affect this hypertension, as shown by Freeman and Page[43] and Heymans et al.[44]

EFFECT OF NEPHRECTOMY IN HYPERTENSION DUE TO UNILATERAL ISCHEMIA AND PYELONEPHRITIS

The successful results of nephrectomy in unilateral pyelonephritis as reported by Butler[45] and others is in keeping with the results of Goldblatt[28] and others who have abolished hypertension of unilateral renal ischemic origin by removal of the ischemic kidney. This important observation should stimulate clinicians to make a careful search for the possibility of unilateral renal disease in hypertensive patients and to give serious consideration to nephrectomy.

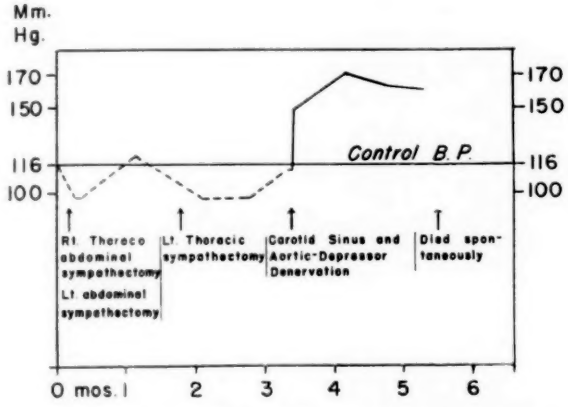

Figure 5. *Effect of Carotid-Sinus and Aortic-Depressor Denervation on the Blood Pressure in a Totally Sympathectomized Dog (Dog 30).*

SUMMARY

Various experimental methods which produce hypertension are discussed.

Corroboration of carotid-sinus denervation and aortic-depressor-nerve section as a means of producing experimental hypertension through increased vasomotor activity is presented.

The production of chronic hypertension by chronic cerebral anemia as an original demonstration is presented.

The nature of experimental hypertension due to renal ischemia is discussed in the light of recent investigations.

Partial sympathectomy, including bilateral splanchnotomy, unilateral adrenalectomy, bilateral lumbar ganglionectomy and even two-thirds to three-fourths sympathectomy, fails to reduce hypertension of carotid-sinus and aortic-depressor-nerve origin.

Preliminary total sympathectomy does not prevent the establishment of moderate hypertension of carotid-sinus and aortic-depressor-nerve origin.

These results in an experimental form of hypertension identical with clinical essential hypertension indicate that bilateral splanchnotomy and ventral rhizotomy lack rational grounds for their use in essential hypertension.

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PELVIC SYMPATHETIC SURGERY FOR THE RELIEF OF BLADDER PAIN*

Resection of the Superior Hypogastric Plexus and Lateral Sacral Sympathetic Ganglia in Tuberculous Cystitis

CARLISLE F. SCHROEDER, M.D.

DETROIT, MICHIGAN

THE purpose of this paper is to give a brief outline of the treatment of intractable pain of tuberculous cystitis by sympathetic denervation of the pelvis. A brief résumé of the history, anatomy and physiology of the nerves involved will be presented in order to give the chronological background.

The superior hypogastric nerve plexus of the pelvis was first described by Winslow[1] in 1732, and has been redescribed and renamed many times since. Jaboulay[2] in 1899 first directed attention toward relief of pain by surgery of the pelvic sympathetic nerves. He used a retrorectal approach which probably destroyed the nerve pathways to or from the hypogastric ganglia, resulting in urinary incontinence, and the operation did not meet with approval. Latarjet[3] in 1913 first adequately described the pelvic sympathetic nerves, but erroneously named the superior hypogastric plexus the "presacral nerve." In 1921 Rochet,[4] attempting to relieve pain in tuberculous cystitis, used periureteral sympathectomies on 3 patients with some relief, but all had an associated incontinence of urine, as did Jaboulay's operated patients, and probably for the same reason. Cotte[5] first performed the operation known as presacral neurectomy in 1925, and has since popularized it with the medical profession, especially gynecologists. Pieri[6] in 1926 attempted to relieve the pain of incurable tuberculous cystitis by the use of Cotte's operation (excision of the superior hypogastric plexus), but was only partially successful. He then did a more radical neurectomy by incising the lateral sacral chains at the level of the first sacral segment, and also cut the rami communicantes to the sacral chain, with better results. In 1932 Learmonth[7] applied Cotte's operation to neurogenic imbalance of the bladder.

Anatomically the nerve supply of the bladder has two subdivisions, the somatic and the autonomic. The latter is composed of the sympathetic and the parasympathetic systems. The somatic nerves take their origin from the anterior division of the third and fourth sacral nerve roots, and carry both motor and sensory fibers to the bladder and posterior urethra. The sympathetic nerves are derived from the thoracolumbar system. Their origin is spread over a large area of the cord; that is, from the celiac, renal and mesenteric abdominovisceral ganglia, as well as from the first four lateral lumbar sympathetic chain ganglia. All these fibers are bottle-necked in the region of the last lumbar vertebral body as the superior hypogastric plexus (presacral nerve). This anatomical peculiarity makes the fibers easily amenable to surgery, even though their origin is so widespread segmentally over the cord. The superior hypogastric plexus divides into two more or less distinct nerve groups; that is, the right and left hypogastric nerves, which carry the thoracolumbar sympathetic fibers to the hypogastric (pelvic) ganglia and hence to the bladder. The superior hypogastric plexus and hypogastric nerves during their course receive branches from other sources: from the last lumbar sympathetic ganglia; from the plexus surrounding the inferior mesenteric vessels, and their continuation; and from the superior hemorrhoidal nerve. They also receive fine filaments from the sacral chain, as shown by our own dissections on fresh cadavers and examinations of living tissue at the time of operation. Anatomists

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From the Department of Urology, Charles Godwin Jennings Hospital, Detroit, Michigan.

using preserved dissection-room cadavers have usually described the plexus as a conglomerate nerve without separation and without anastomosing fibers. Experimentally, on animals, and by stimulation during operation on human beings, it has been shown that the thoracolumbar nerves supply the region of the trigone more exclusively with fibers to the hypogastric (pelvic) ganglia. As they emerge from these, the fibers can be divided again into three groups, the upper root supplying the fundus of the bladder, the middle root supplying the midportion of the bladder, and the lower root going to the lower aspect of the bladder, the vesical neck and the adjacent urethra.

Table 1. *Reported Cases of Tuberculosis of the Bladder Treated by Sympathetic Surgery.*

CASE NO.	AUTHOR	REMARKS	OPERATION	RESULTS
1	Learmonth[7]	Age 33	Presacral neurectomy	Marked relief of pain; only slight frequency.
2	Learmonth[11]	Age 45; tuberculosis of genitourinary tract for 5 years.	Cutting of efferent branches of hypogastric ganglia and presacral neurectomy	Died postoperatively
3		Age 38; tuberculosis for 6 years; nephrectomy.	Presacral neurectomy	Condition good 6 months; then return of symptoms.
4	Learmonth and Braasch[12]	Age 35 (woman); 5 years of urinary frequency and severe pain.	Presacral neurectomy	Disappearance of pain; persistence of frequency.
5	Rochet[4]	Unilateral crisis type of pain	Periureteral sympathectomy (unilateral)	Relieved unilateral crises until death in 4 months
6		Pain originating from ureteral stump	Periureteral sympathectomy (unilateral)	Lived 2 years; relief.
7		Bilateral type of crisis pain	Periureteral sympathectomy (bilateral)	Complete relief
8	Van Duzen[13]	Genitourinary tuberculosis	Presacral neurectomy	Persistent pain and incontinence
9	Perrin[14]	Severe dysuria	Presacral neurectomy	Immediate amelioration of frequency; died 3 months postoperatively.
10		Severe pain and frequency	Presacral neurectomy	No pain; died 15 days postoperatively.
11		Frequency to point of incontinence; severe dysuria.	Presacral neurectomy	Amelioration of frequency
12	Pieri[15]	Age 40; nephrectomy for tuberculosis.	Presacral and hypogastric neurectomy	Some relief of pain for 4 years
13		Age 16; severe pain and frequency.	Presacral neurectomy; division of sympathetic chain and rami communicantes.	Excellent relief of pain 2 years until death
14		Age 36; tuberculosis of lungs and urinary tract; pain and frequency.	Presacral neurectomy; division of sympathetic chain and rami communicantes.	Died in 4 days of uremia; apparent relief of pain.
15	Reynard and Michon[16]	Operated on five times for tuberculosis of urinary tract	Presacral and hypogastric neurectomy	Relief only after resection of ulcer in bladder
16	von Suermondt-Leiden[17]	No history	Presacral neurectomy	Less frequency
17		No history	Presacral neurectomy	Less frequency
18		No history	Presacral neurectomy	Less frequency
19	Kwan, Char and Tung[18]	Severe pain and frequency	Presacral neurectomy	Symptomatic improvement
20	von Noszkay[10]	Bladder pain, frequency and diminished capacity.	Presacral neurectomy	Some pain remained; tenesmus diminished; capacity increased.
21		Bladder pain, frequency and diminished capacity.	Presacral neurectomy	Favorable
22		Bladder pain, frequency and diminished capacity.	Presacral neurectomy	Favorable
23		Bladder pain, frequency and diminished capacity.	Presacral neurectomy	Favorable
24		Bladder pain, frequency and diminished capacity.	Presacral neurectomy	Favorable
25		Bladder pain, frequency and diminished capacity.	Presacral neurectomy	Unfavorable

than they do the remainder of the bladder, a point which may be important in the selection of operative cases when considering all types of bladder pain.

The parasympathetic fibers (craniosacral outflow) from the anterior primary divisions of the second, third and fourth sacral nerves are divided into three so-called pelvic nerves and course

The physiology of the nerves supplying the bladder is the subject of considerable controversy at the present time, from the point of view both of clinical observation and of animal experimentation. Learmonth and Braasch[8] found that by arranging cystoscopic examinations at the time of operation stimulation of the sympathetic nerves brought about "closure of the ureterovesical orifices, con-

traction of the trigone, closure of the internal sphincter, contraction of the prostate, seminal vesicles and ducts, inhibition of the expulsive power of the bladder; and that they carried afferent fibers for sensation of pain in the bladder." Recent work by other observers, especially McCrae and Macdonald,[9] raises doubt whether either system is exclusively excitor or inhibitor in function or whether they are antagonistic, but suggests that they do work together, along with the somatic nerves, in the regulation of bladder function. The parasympathetic fibers are apparently the most important and carry the stronger impulses.

With the reports of cases operated on for the relief of bladder pain in tuberculous cystitis, as summarized in Table 1, and with the foregoing anatomical facts and physiological observations in mind, we attempted to improve the results, and planned a more radical sympathectomy than Cotte's presacral resection and Pieri's addition of section of the sacral chains and rami communicantes. To accomplish this, we excised the superior hypogastric plexus, as did Cotte, sectioned the lateral sacral chains, as did Pieri, and then performed an exeresis of these lateral sacral chains. Just as the operation of phrenico-exeresis as proposed by Felix[10] causes a more marked and permanent paralysis of the diaphragm, so this operation results in a more marked and permanent relief of pain in the bladder. We still leave intact, however, the parasympathetic fibers, as well as the pudendal somatic supply. In order to remove the sensory fibers of both these groups, and not the motor fibers, which has always been done with nerve resections resulting in incontinence (Jaboulay[2] and Rochet[4]), we have added, when necessary, the postoperative use of intrathecal alcohol; with exacting control of the subarachnoid dispersion of the alcohol, the injection results in no motor paralysis, but does give complete relief of pain.

It has previously been pointed out that fibers which comprise the superior hypogastric plexus are diffused over a large area of the cord, and are therefore not well adapted to treatment with intrathecal alcohol; yet they are readily accessible to the surgeon, since anatomically they are converged in a small, easily accessible space.

Intrathecal alcohol therapy though simple is not without danger, and numerous accidents and sequelae have been recorded; it should be used only as a last resort. Its greatest danger is its relative simplicity. A spinal tap is carried out as low down as possible, with the patient in the lateral prone position. The hips are then elevated on firm pillows, or, better, by lowering the head end of the flat supporting surface, and the torso is rotated anteriorly into an oblique position, since only the most caudal sensory (posterior) nerve components are to be primarily affected. One half to 1 cc. of absolute alcohol is slowly injected. The needle is removed, and the patient is kept in this exaggerated position for three hours and in a prone position for three more. A lapse of one or two weeks is allowed between injections; that is, before intrathecal alcohol is again used, with the opposite side uppermost.

The technic of simple excision of the superior hypogastric plexus has been frequently given in the literature and will not be repeated here, except to state that it must be complete. It is a good rule to consider in surgery that in all cases the group consists of a plexus rather than a nerve, so that the smallest fiber is not overlooked and left undivided at operation. The technic of resection of the lateral sacral chains is not difficult and can be approached through the same incisions. A lower abdominal midline incision is carried out and the peritoneum opened. The entire bowel except the lower sigmoid is pushed into the upper abdomen, so that the inverted V made by the branches of the common iliac vessels is in view. Identification of the contents of the mesentery of the sigmoid is important, both for nerve fibers to and from the superior hypogastric plexus, and to avoid injury to the inferior mesenteric vessels. The posterior peritoneum is opened longitudinally in the mid-line over the bifurcation of the great vessels for a distance of 7 to 10 cm. down into the concavity of the sacrum. The superior hypogastric plexus is identified and dissected well down, so that the hypogastric nerves are exposed, and all fibers leading to and from the group are disrupted, dissection being aided with nerve hooks. The nerve group is next excised, and the cut ends are tied or silver-clipped. The posterior peritoneal flaps are dissected further laterally to a point just lateral to the anterior sacral foramina. The latter are important landmarks, since the sacral sympathetic chains run just medial to the foramina and can be easily felt and "snapped" between the finger and sacrum, as is the vas deferens between the fingers and thumb. The chain is then incised above the first sacral segment, and the distal cut end is grasped with a clamp, which is twisted, curling the nerve chain over its end; at the same time the chain is pulled out. With a blunt dissector the chain is freed simultaneously of all its connections so that it can be pulled out more readily. Two or three ganglia are obtained and bleeding is scant, being easily controlled with packs, which are re-

moved before the posterior peritoneum is closed. The abdomen is closed without drainage.

CASE REPORTS

Case 1. M. D., a 12-year-old girl, gave a 6-year history of urinary tuberculosis, confirmed by guinea-pig inoculation. There was voiding every half hour, with incontinence at night. The bladder capacity was 80 cc. Excision of the superior hypogastric plexus and lateral sacral chains was performed July 3, 1934. The bladder capacity

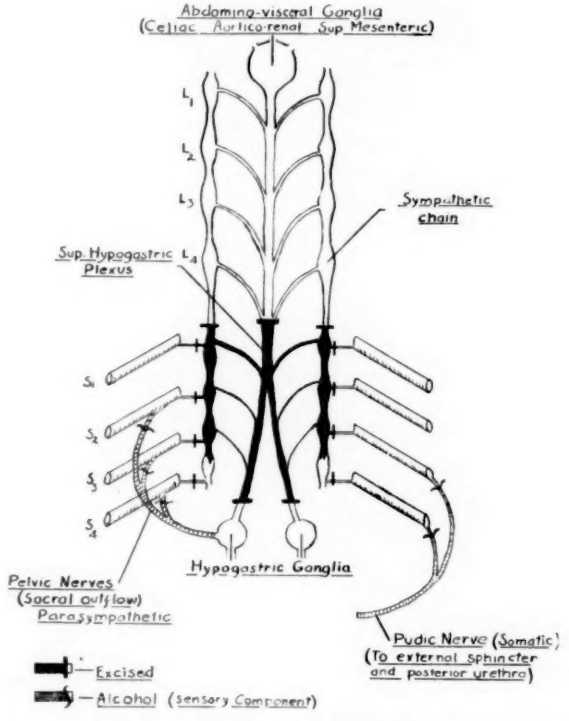

Figure 1. *Excision of Superior Hypogastric Plexus, Exeresis of Sacral Sympathetic Chains and Intrathecal Alcohol Injection.*

increased to 180 cc., with no pain or incontinence. There was voiding every 2 hours. The patient was able to attend school for the first time in 6 years.

Case 2. A. F., a 26-year-old man, gave a 3-year history of symptoms of genitourinary tuberculosis. There were bilateral destructive lesions of the kidneys. A guinea-pig inoculation of urine from the bladder was positive. The bladder capacity was 90 cc., and there was voiding every half hour. Resection of the superior hypogastric plexus and right lateral sacral chain was done in October, 1934. Relief of pain followed and frequency ceased. The bladder capacity was increased to 175 cc., without pain, and the patient was able to work as an apprentice printer.

Case 3. J. C., an 18-year-old girl, gave a long history of bone and joint tuberculosis. There was a 15-month history of severe dysuria and frequency, during which the patient had led a "catheter life" in a sanatorium. Cystoscopy with pyelograms and guinea-pig inoculation revealed bilateral tuberculosis. Resection of the superior hypogastric plexus and lateral sacral chains was performed in November, 1934. There was complete relief of pain; the bladder capacity increased to 290 cc. and continued at 300 cc.

Case 4. R. B., a 42-year-old man, had a 24-year history of bone and joint tuberculosis, with a diagnosis of genitourinary tuberculosis 7 years previously. The patient had voided every 15 minutes with severe pain for the last 2 months. Guinea-pig inoculations were positive. Resection of the superior hypogastric plexus and lateral sacral chains was done in November, 1934. There was no further pain; the patient voided every 2½ or 3 hours, with a bladder capacity of 175 cc.

Case 5. A. B., a 48-year-old man, 4 years previous to examination had had painful and frequent urination; 10 months previously hematuria had developed, followed by bilateral tuberculous epididymitis and increased bladder symptoms, so that he voided every 15 minutes, with a bladder capacity of 100 cc. A diagnosis of renal tuberculosis was made and was confirmed by guinea-pig inoculation. Excision of the superior hypogastric plexus and the lateral sacral chains was followed by a rise in bladder capacity to 275 cc., with frequency decreased to every 2 or 3 hours.

Case 6. A. G., a 26-year-old woman, underwent a nephrectomy for tuberculosis in February, 1934, with no relief of bladder symptoms. Frequency and dysuria persisted in spite of hysterectomy for fibroids. There was voiding every 5 to 15 minutes. Excision of the superior hypogastric plexus and left lateral sacral chain was done in December, 1935. The bladder capacity rose to 300 cc., but the same pain persisted and there were periods during which the frequency recurred. Intrathecal alcohol was injected in April, 1936. This was followed by complete relief of pain and of frequency. The patient now voids every 3 to 4 hours during the day and twice each night.

Case 7. R. M., a 32-year-old woman, underwent a nephrectomy for tuberculosis in 1930, with some relief of bladder symptoms for 6 years. This was followed by recurrence, the symptoms becoming unbearable. Tuberculosis was found in the remaining kidney. There was voiding every 15 or 20 minutes, with pain referred to the urethra. Excision of the superior hypogastric plexus and the lateral sacral chains was done in September, 1937, with relief of pain except that referred to the urethra. Injection of intrathecal alcohol in February, 1936, was followed by complete relief. The bladder capacity increased to 400 cc., and frequency decreased to every 3 or 4 hours.

COMMENT

We have delayed reporting this series of cases until the results seemed relatively permanent. Relief of frequency following pelvic sympathectomy is at times greater than the increased bladder capacity, showing decreased bladder irritability, as well as relief of pain. Healing of the bladder lesions following operation may be coincident with the increased blood supply and tissue nutrition associated with postoperative vasodilatation. Relief of pain has been uniformly successful, especially where lesions are confined to the trigone. (Tuberculous bladder lesions are most frequently found about the ureteral orifices and trigone.) It has been noted in our cases of bladder pain due to other conditions than tuberculosis that results are

less striking, especially in cases of interstitial cystitis where the lesions are not on the trigone. This brings up again the difference in the anatomical distribution of the nerves, as stated earlier; in such cases, excision of the superior hypogastric plexus is not sufficient to bring about relief of pain. We are now using radical excision of the pelvic sympathetics along with the intrathecal injection of alcohol in all cases of interstitial cystitis, but the series is not large enough nor the individual cases of long enough duration to permit the drawing of conclusions. The results are, however, very gratifying.

CONCLUSIONS

Sympathetic surgery for the relief of bladder pain in incurable tuberculous cystitis is a rational procedure.

The results of excision of the superior hypogastric plexus and lateral sacral chains show significant and lasting improvement, in contradistinction to cases with less complete denervation reported in the literature.

The intrathecal injection of alcohol, with controlled subarachnoid dispersion, in connection with surgery gives a complete sensory denervation, without incontinence.

7815 East Jefferson Avenue.

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VARIOUS METHODS OF DETERMINING THE EARLY DIAGNOSIS OF ARTERIOSCLEROSIS IN DIABETES*

LOUIS I. KRAMER, M.D.†

PROVIDENCE, RHODE ISLAND

IT IS evident from the literature that the chief cause of death in diabetes has shifted from coma to arteriosclerosis. Joslin[1] reports an increase in the death rate from vascular disease, since the advent of insulin, from 28 to 47 per cent, and in a more recent study he[2] asserts that in a series of 42 deaths in cases of diabetes none were attributed to coma, but 50 per cent were due to vascular disease. One reason for this change is the fact that the diabetic patient's life expectancy has been increased, so that he is likely to live long enough to develop normal physiopathologic changes including arterial changes. Unfortunately there is a much graver reason — an unknown factor which seems to be inherent in the patient with diabetes, making the vessels vulnerable to arterial changes in the young as well as in the old. According to Allen,[3] "Arteriosclerosis is present in every patient with diabetes past middle life, and at any age, according to others, provided the disease has been present for ten years or longer." Autopsies on 52 of Joslin's[1] cases showed arteriosclerosis in 19 persons who had had the disease for more than five years. Wilder[4] in 81 autopsies on diabetic patients found an extremely high incidence of arteriosclerosis, and Warren[5] makes this striking statement: "I have yet to see at autopsy a diabetic, or to read a protocol of a diabetic, whose disease has lasted five years or more, free from arteriosclerosis, regardless of age." Rabinowitch, Ritchie and McKee[6] in a recent report of 500 carefully studied cases of diabetes, found an incidence of 62.6 per cent of cardiovascular disease; when the cases were grouped according to age, cardiovascular disease was evident in 54.7 per cent at the age of fifty or under.

Table 1 illustrates strikingly the rapidly shifting

*From the Diabetic Clinic, Rhode Island Hospital, Providence.

†Visiting physician, Rhode Island Hospital, Providence.

cause of death in diabetes from coma to arteriosclerosis.[7] Of 342 deaths reported during the Naunyn era, from 1894 to 1914, 15 per cent were caused by arteriosclerosis in some form and 61 per cent by coma. Of 805 deaths reported during the Allen era, from 1914 to 1922, 26 per cent were attributed to arteriosclerosis and 42 per cent to coma. Since the discovery of insulin in 1922 (Banting era), of the 979 deaths recorded up to 1929, 44 per cent came from arteriosclerosis and 17 per cent from coma; of a total of 474 deaths from 1926 to 1929, 48 per cent were traceable to arteriosclerosis and only 11 per cent to coma.

Table 1. *Percentages of Deaths in Diabetes Due to Arteriosclerosis and to Diabetic Coma in Each of the Important Eras of Treatment.*[7]

ERA	TOTAL DEATHS	DEATHS FROM ARTERIOSCLEROSIS	DEATHS FROM COMA	AVERAGE DURATION OF DIABETES
		%	%	yr.
Naunyn — 1894 to 1914	342	15	61	4.7
Allen — 1914 to 1922	805	26	42	5.4
Banting — 1922 to 1929	979	44	17	8.1
1922 to 1926	505	40	22	7.6
1926 to 1929	474	48	11	8.7

The evidence from all sources points obviously to arteriosclerosis as the real menace threatening the diabetic patient, but why he should be more vulnerable to cardiovascular changes than a person without the disease is still unknown. However, investigators have for some time suspected that improper fat metabolism is of sufficient importance to merit thorough study, and there is ample evidence justifying that suspicion.

The thick and milky appearance of blood drawn from a patient with diabetes was a common observation when bloodletting was in vogue. Its significance, however, aroused very little comment, and was obviously lost sight of until Fischer[8] in 1903 made a similar observation. He concluded that the blood lipoids were elevated in diabetes. Bloor,[9] after studying a group of cases from Joslin's clinic, corroborated Fischer's contention. A similar study by Gray[10] on 171 patients revealed very high blood-fat values in 78 per cent of the cases, and after correlating the high-fat values with the duration of life in the fatal cases he concluded that they were of grave prognostic significance. Today, such a gloomy outlook is not justified because of the stabilizing effect of insulin on blood cholesterol.

Rabinowitch[11] believes that there is a definite relation between high blood-lipoid values and cardiovascular disease, especially among younger diabetic patients. His study revealed that those who showed evidence of cardiovascular damage invariably had high blood-cholesterol values. Recently, Leary[12] confirmed the observations of earlier investigators.[13, 14] By feeding cholesterol to rabbits he succeeded in producing lesions simulating human atherosclerosis, and he regards this phenomenon as a specific disorder of the cholesterol metabolism.

Obviously, any study which deals with the etiology of arteriosclerosis should include blood-cholesterol determinations. It is interesting to contrast the blood-lipoid values as reported by investigators[9, 10] prior to the insulin era with those reported since the use of insulin and better diets.[15, 16] Already there are indications, or at least assumptions, that the incidence of arteriosclerosis is on the decline. Shepardson[17] believes that the reduced incidence parallels the reduction of lipemia since the advent of insulin therapy.

For the early diagnosis of arteriosclerosis, however, no one method is sufficient. The manifestations are varied and many. The detection of cardiovascular disease is best studied from various angles. A roentgenological study of the cardiovascular system is very helpful. Bowen and Koenig[18] have been able to demonstrate vascular changes in the lower extremities in 63 per cent of patients beyond the age of forty. In a similar study by Morrison and Bogan,[19] a total of 162 observations revealed the following interesting fact: the longer the duration of the diabetes the greater the incidence of arteriosclerosis. Forty per cent of those who had had diabetes for five years showed evidence of arteriosclerosis, and the percentages for the ten-, fifteen- and twenty-year groups were 50, 83 and 92 respectively.

For additional methods the reader is referred to papers by other authors. The procedures for studying the degree of vascular disease are, singly or in combination, the oscillometer, the response to a cutaneous injection of a diluted solution of histamine, palpation of the pulsations of the dorsalis pedis and posterior tibial arteries and examination of the fundi and retinal vessels.

In this report an attempt is made to evaluate the merits of the various tests and to ascertain the virtue of supplementing one with another. One hundred cases selected at random from the Diabetic Clinic at the Rhode Island Hospital were studied. Blood-sugar and blood-cholesterol determinations were made in each case. The extremities were x-rayed and the degree of calcification was noted. The fundi, especially the retinal vessels, were studied and for the sake of uniformity were all examined by the same physician, Dr. Harry C. Messinger. The pulsations of the dorsalis pedis and posterior tibial arteries were recorded and the degree of pulsation was noted as absent, +, ++, +++ and ++++. Oscillo-

metric tracings were taken and cutaneous histamine reactions observed in all suspected cases. The scheme adopted for the study of each case was as follows:

1. Name and age
2. Eyes
   a. Intraocular inflammation
   b. Cataract
   c. Eye muscles
   d. Fundi, especially retinal vessels
3. Duration of diabetes
   a. History
   b. Length of time in clinic
4. Blood pressure
5. Blood sugar
6. Blood cholesterol
7. Dorsalis pedis pulsation
   a. Right
   b. Left
8. Posterior tibial pulsation
   a. Right
   b. Left
9. X-ray study of lower extremities for evidence of arterial calcification
10. Oscillometric tracing on all cases showing either calcification by roentgenography or poor peripheral circulation by palpation, or both
11. Histamine cutaneous test in all cases showing pathologic changes in lower extremities.

An analysis of this study, taking the group of cases as a whole, reveals a 38 per cent incidence of arteriosclerosis. However, when the cases are classed by decades of life one readily notes an increase in the incidence with each decade (Table 2). There are no cases in the first and second decades. In the third decade the incidence is 8 per cent, in the fourth 17 per cent, and so on until we reach the ninth decade, where the incidence is 100 per cent. It would be irrational to consider diabetes the sole offender in this climbing incidence. Undoubtedly a considerable portion of vascular disease can be and should be attributed to normal degenerative changes relevant to age.

The question of hypertension in diabetes and its relation to arteriosclerosis is also of interest. Are the two parallel? Only 18 per cent of this entire group gave a systolic-pressure reading higher than 150, and, interestingly enough, in no patient under thirty was the blood pressure above normal. However, 11 (28 per cent) of those showing evidence of sclerosis had hypertension.

Does an abnormal blood sugar forecast the early development of arteriosclerosis? In the opinion of Mosenthal,[25] hyperglycemia is in no way a causative factor; and this is confirmed by our study. Ninety-two per cent of the group gave blood-sugar readings greater than 120 mg. per cent (the accepted normal), while of the patients with arteriosclerosis only 32 (85 per cent) had blood-sugar values higher than 120 mg.

Some observers[11] have stated that there is distinct relation between blood lipids and arteriosclerosis. The cholesterol studies in this group do not warrant such a conclusion. Only 23 per cent of the total cases, and only 8 (21 per cent) of the arteriosclerotic cases, gave readings higher than 225 mg. per cent (the accepted normal). The discrepancy between the conclusions of other observers and the findings in this group may be attributed to our use of insulin, and to the change from a high-fat, low-carbohydrate diet to a low-fat and high-carbohydrate one. Insulin seems to exert a stabilizing effect on fat metabolism. The cholesterol values have not been nearly so high since the use of insulin and of diets approaching normal.

Deposition of calcium in the vessel walls indicates vascular disease. Roentgenologically this can be demonstrated very adequately. X-ray studies of the lower extremities were made on all our patients. Twenty-four per cent of the entire group showed evidence of sclerosis as compared with 63 per cent in the arteriosclerotic group (Table 3). However, advanced sclerosis may exist without one's being able to demonstrate such deposition roentgenologically, as evidenced by the 14 cases with negative x-ray findings but with other indications of vascular damage.

The pulsations of the dorsalis pedis and pos-

Table 2. *Clinical and Laboratory Findings in Entire Group, Arranged by Decades.*

Decade	No. of Cases	Duration of Diabetes		Blood Pressure			Blood Sugar			Blood Cholesterol			Evidence of Arteriosclerosis	
		Average	Range	High	Low	Average	High	Low	Average	High	Low	Average	No.	Per Cent
		yr.	*yr.*	*mm.*	*mm.*	*mm.*	*mg.*	*mg.*	*mg.*	*mg.*	*mg.*	*mg.*		
First	1	7		75/40			182			187				
Second	16	4.0	½ to 7	134/80	82/50	107/63	480	190	300.7	280	157	229.1		
Third	12	5.7	½ to 14½	130/85	110/70	116/73	400	80	238	272	100	181.5	1	8
Fourth	12	5.5	½ to 12	180/105	100/80	119/79	334	112	203.7	286	94	206.25	2	17
Fifth	14	4.8	½ to 11	190/98	110/65	134/77	298	89	163.5	240	130	195	6	43
Sixth	23	3.4	½ to 12	200/120	110/70	142/83	364	121	190.5	300	133	197	11	48
Seventh	17	4	½ to 17	220/80	130/80	153/84	236	82	168	300	164	203.6	13	77
Eighth	5	9	5 to 14	180/108	100/80	164/79	200	115	152.4	200	182	194	5	100

terior tibial arteries are important objectives when searching for information regarding the circulation in the lower extremities. A good dorsalis pedis pulse was observed in 25 (66 per cent) of the arteriosclerotic group; the pulse was diminished in 6 cases and absent in 7. A good posterior tibial pulse was noted in 11 cases (29 per cent); the pulse was diminished in 6 cases and absent in 21. Does a good pulse rule out arteriosclerosis? Obviously not. Sixteen (66 per cent) of the 24 cases showing calcium deposition in the vessel walls by x-ray had either a good dorsalis pedis pulse or a good posterior tibial pulse, or both. Again, curiously enough, in 3 cases with no pulsation the roentgen-ray findings were negative.

In the study of the eyes no incidence of intraocular inflammation was noted and in only 1 patient was muscular abnormality found. The case report follows:

H.D., 46 years old, had had diabetes for 3 years. The blood pressure was 130/80, the blood sugar 118 mg. per cent and the blood cholesterol 200 mg. per cent. No calcification was shown by x-ray, and both the dorsalis pedis and posterior tibial pulsations were good.

The eye findings were as follows: "The pupils are equal, regular and react to light and accommodation. The motility of the right eye is limited in all directions except outward and upward, the greatest limitation being inward. The eyes are prominent and the right fissure is slightly larger than the left. There seems to be a myasthenia of the extraocular muscles, most marked in the right eye, and especially in the right internal rectus. There are no retinal changes."

This patient had a unilateral thyroidectomy 3 years prior to this study and his present basal metabolic rate is +34 per cent. He shows no evidence of arteriosclerosis.

Five patients showed evidence of cataract formation: 1 in the fifth decade, 3 in the seventh and 1 in the eighth. Twenty-three per cent of the entire group, or 60 per cent of the arteriosclerotic group, had retinal lesions: 1 in the third decade, 4 in the fifth, 6 in the sixth, 9 in the seventh and 5 in the eighth. Here, too, one is at a loss as to how much to blame the diabetes and how much the advancing age. Only 4 of the patients under fifty showed retinal changes, as compared with 19 of those aged fifty and over. These findings compare favorably with those of Spalding and Curtis,[26] who found a 20 per cent incidence of arteriosclerosis on ophthalmoscopic examination of the eye grounds.

The arteriosclerotic group was studied for possible detection of deficient circulation of the lower extremities with the oscillometer and the histamine intracutaneous test. The Pachon recording oscillometer was employed, and the readings at the ankle joint were recorded at various pressure levels. The height of the oscillations was used in grading the degree of efficiency of the circulation. With an oscillation of 5 mm. or over the circulation was considered good or adequate, with 2 to 4 mm. as fair, with 1 mm. as poor, with no response as zero. Fifteen cases (39 per cent) showed normal oscillogram tracings; in 7 (18 per cent) the tracings were considered fair; 15 (39 per cent) gave a poor reading, and in 1 case the reading was zero.

How may one interpret with impunity the low incidence of circulatory impairment and the high incidence of relatively normal readings? Kramer[20] supplies a satisfactory answer when he says, "Since the mechanism of the oscillometer is so arranged as to give us information upon the gross blood flow and the rhythmic expansion and contraction of the vessels, it is only fair to assume that if a

Table 3. *Findings in the Arteriosclerotic Group, Arranged by Decades.*

Decade	No. of Cases	Average Blood Pressure	Average Blood Sugar	Average Blood Cholesterol	X-Ray Findings	Pulsations		Eyes		Oscillometer Reading	Histamine Response
						Dorsalis pedis	Posterior tibial	Fundi	Cataract		
		mm.	*mg. %*	*mg. %*							
Third	1	120/70	298	160	Positive	3+	2+	Sclerosis		Good	Good
Fourth	2	115/80	280	205	1 Positive 1 Negative	4+ 3+	1, 4+ 1, 3+	2 Normal		1 Good 1 Fair	2 Good
Fifth	6	126/76	198	194	4 Positive 2 Negative	3, 4+ 2, 3+ 1, 2+	2, 3+ 2, 2+ 2, 0	4 Normal 2 Sclerosis	1	5 Good 1 Poor	4 Good 2 Fair
Sixth	11	138/76	177	203	9 Positive 2 Negative	2, 4+ 3, 3+ 3, 2+ 1, 1+ 2, 0	1, 2+ 4, 1+ 6, 0	5 Normal 6 Sclerosis		4 Good 2 Fair 4 Poor 1 0	8 Good 1 Fair 2 0
Seventh	13	155/85	157	207	7 Positive 6 Negative	2, 4+ 2, 3+ 4, 2+ 2, 1+ 3, 0	2, 3+ 1, 2+ 2, 1+ 8, 0	4 Normal 9 Sclerosis	3	4 Good 3 Fair 6 Poor	7 Good 2 Poor 4 Not done
Eighth	5	164/92	152	194	2 Positive 3 Negative	3, 1+ 2, 0	5, 0	5 Sclerosis	1	1 Fair 4 Poor	1 Good 2 Fair 2 Poor
Cases showing evidence of arteriosclerosis	38	18	32	8	24	13	27	23	5	16	6

satisfactory collateral circulation has been established we may get normal readings despite the presence of definite disease in some of the vessels." The presence of vascular damage does not necessarily imply that the circulation is not patent.

The histamine intracutaneous test was carried out on 34 of the arteriosclerotic patients. One tenth of one cubic centimeter of a 1:1000 solution was injected intracutaneously and the response was noted at five-, ten- and fifteen-minute intervals. The sites selected were those above and below the knee, above the ankle and on the dorsum of the foot. Normally a flare and a wheal appear at the end of five minutes and usually become more marked at the time of the ten-minute observation. At the end of fifteen minutes, if no local reaction is observed the response is considered negative. In 23 cases (67 per cent) the reaction was normal. Six (17 per cent) showed a delayed response. In 4 (12 per cent) the reaction was minimal and in 2 (6 per cent) it was absent. All the patients except 2 who showed a normal response to histamine had a normal (good) oscillometric tracing. Here again the high incidence of a normal response does not discredit the test. On the contrary, the test gives one information as to the patency of the capillaries and peripheral circulation and indirectly tells one something about the status of the deep and large vessels.

Table 3 summarizes the positive findings in the arteriosclerotic group. It will be observed that by a combination of methods the incidence is 38 per cent. It is also of interest to note that the number of cases which showed hypertension parallels fairly well the numbers which showed positive x-ray findings and retinal arteriosclerosis — 18, 24 and 23 per cent respectively. A good pulsation means an efficient circulation, but by no means indicates absence of vascular disease. However, when no pulse is present and in addition one gets a negative oscillometer reading and a poor histamine response, it is safe to assume that vascular lesions are present and that the circulation is impaired. Interestingly enough, the 13 patients with poor or no pulsation of the dorsalis pedis artery compare favorably with the 16 who showed a poor oscillometric index. The histamine response was negative or poor in 6 cases. The pulse of the posterior tibial artery was at least only poorly palpated in 27, in only 8 were the lipoid values above normal, while high blood-sugar values were found in 32.

CONCLUSIONS

A multiplicity of methods yields a far greater incidence of arteriosclerosis than does any one single method.

The blood-cholesterol values have decreased to a relatively normal level since the advent of insulin and the use of diets containing a high-carbohydrate and low-fat content.

The number of patients having a poor or no pulsation of the peripheral vessels of the lower extremities compares favorably with the number showing a poor oscillometric index.

The histamine intracutaneous response is the most reliable guide in determining the presence or absence of a sufficient collateral circulation.

The ophthalmological examination of the fundi, the x-ray study of the lower extremities and the blood-pressure readings demonstrate a relatively equal incidence of arteriosclerosis.

126 Waterman Street.

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ALCOHOL TOLERANCE TESTS IN NORMAL INDIVIDUALS AND IN PATIENTS WITH DIABETES MELLITUS AND DIABETES INSIPIDUS*

The Effect of Pituitrin, Insulin, Food and Forced Water on Blood and Urine Alcohol Levels After The Ingestion of Alcohol

HARRY BLOTNER, M.D.†

BOSTON

ALCOHOL is absorbed with extreme rapidity from the gastrointestinal tract, even though it destroys digestive enzymes[1] and prevents or delays the proper digestion of food.[2] Furthermore, Mellanby[3] and others have shown that the metabolism of alcohol, regardless of the amount present, proceeds at a constant rate for the individual. There are very few conditions which are known to modify the rate of absorption or metabolism of alcohol. For example, insulin has been said by some to increase the rate of disappearance of alcohol from the blood, whereas food was thought to slow the rate of its absorption.

Normal people have a varying tolerance to alcohol, just as they do to tobacco and other drugs. A few patients with diabetes insipidus have told me that they can tolerate liquor better than normal people. Frequently patients with diabetes mellitus ask whether a drink of alcohol would do them harm. To investigate these points appeared to be of practical importance.

This paper presents a study of alcohol tolerance tests in normal and diabetic persons, and of the effect of pituitrin, insulin, food and forced water intake on these tests.

METHODS

The alcohol tolerance was determined in much the same way that glucose tolerance is ordinarily studied. A dose of 0.6 cc. of absolute alcohol per kilogram of body weight was employed as a test meal. The standard solution contained 50 cc. absolute alcohol, 50 cc. grapejuice and 150 cc. water. This was a reasonably palatable mixture and never caused vomiting, nausea or diarrhea. It was chilled and administered to the patients in the morning after fasting overnight. The alcohol concentration was determined in simultaneous samples of venous blood and urine which were obtained before the test meal and at half-hour intervals for four hours thereafter. The dose of absolute alcohol usually varied from 40 to 50 cc. The patients remained in the laboratory during the test period without additional food, but with some added water if desired. The symptoms of intoxication were noted.

The chemical method at first used for the determination of alcohol was that described by Fleming and Stotz.[4] It is a modification of that employed by Nicloux[5] for use of the Folin–Wu blood filtrate, and is based on the principle of the reduction of potassium dichromate in sulfuric acid, with the estimation of the excess by ferrous ammonium sulfate and titration with potassium permanganate. Chiefly because the potassium permanganate solution is unstable, and because to adjust it to proper strength often requires considerable time, the method was modified further in this clinic[6] to determine the concentration of alcohol by the Evelyn photoelectric colorimeter.[7] This measures directly the diminution of the color of the acid bichromate solution due to reduction by alcohol, and eliminates the potassium permanganate and ferrous ammonium sulfate solutions and the titration. The results obtained are accurate and agree with those obtained with the titration method.[6, 8]

The analyses were made on 10 cc. of the Folin-Wu blood filtrate and on 10 cc. and 1 cc. of the urine obtained before and after alcohol ingestion respectively.

ALCOHOL TOLERANCE CURVES IN NORMAL PEOPLE

The alcohol tolerance was studied, first, in 10 normal persons in order to obtain a standard with which to compare the tolerances observed in diabetic patients. The average urine and blood values are given in Chart 1. The fasting blood alcohol varied from 0 to 7 mg. per cent. After the ingestion of alcohol there was a rise in the curve, which reached its average maximum concentration of 41 mg. in one hour and decreased gradually to a level of approximately 15 mg. in four hours.

The fasting urine alcohol varied from 0.4 to 2 mg. per cent. After the ingestion of alcohol it rose to an average maximum concentration of 50 mg. in one and a half hours, and decreased in four hours to an average level of 16 mg. This concentration was appreciably lower than that in the blood at the first half hour, but exceeded it at

*From the Medical Clinic of the Peter Bent Brigham Hospital, Boston.
†Associate in medicine, Peter Bent Brigham Hospital, Boston.

one hour and one and a half hours. Also the blood alcohol, during its period of decline, was slightly lower than the urine alcohol. Smith and Stewart[9] surmised that this relation between blood and urine alcohol was to be expected.

It was interesting to observe that some symptoms of intoxication appeared in these individuals, in spite of the fact that the blood-alcohol concentrations were less than 63 mg. per cent. These symptoms included being exhilarated, euphoric, talkative, slightly dizzy and unstable. The findings were in striking contrast to those observed by Selesnick,[10] who noted that the clinical manifestations of intoxication did not appear in alcoholic patients at the Boston City Hospital until the blood alcohol exceeded 200 mg. per cent. It is quite likely that this marked difference is due to the fact that Selesnick's patients were chronic alcoholics who could tolerate a much higher blood alcohol level through habituation than could normal persons. On the other hand, Heise[11] found that loss of efficiency and impairment of judgment occurred even when the blood and urine contained as little as 20 mg. per cent of alcohol.

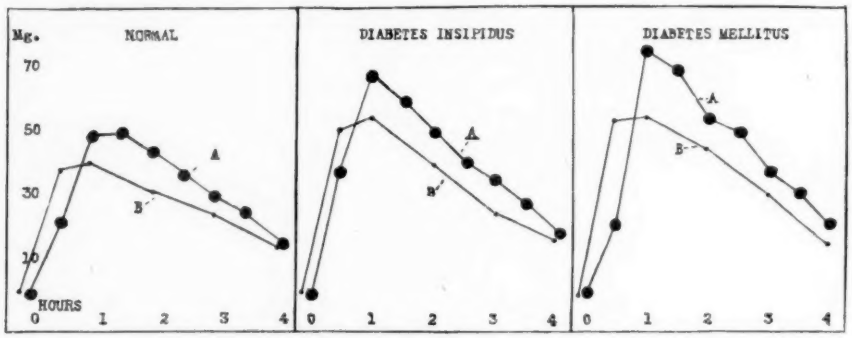

Chart 1. *Response to Ingestion of Alcohol.*

Average curves for urine (A) *and blood* (B) *alcoholic contents obtained in 10 normal people, 5 with diabetes insipidus and 6 with diabetes mellitus, after the ingestion of 0.6 cc. of absolute alcohol per kilogram of body weight. In this and subsequent charts the alcohol is recorded in milligrams per cent.*

ALCOHOL TOLERANCE IN DIABETES INSIPIDUS, WITH AND WITHOUT PITUITRIN THERAPY

Alcohol tolerance tests were obtained in 5 patients with diabetes insipidus after pituitrin had been omitted for a few days and the fluid intake and output had been markedly increased (Chart 1). The tests were repeated after the patients had received pituitrin for several days, when the fluid intake and output were normal. The morning of the day of this test, 1 cc. of obstetrical pituitrin was administered intranasally on a pledget of cotton immediately after the imbibing of alcohol. In 2 patients the tests were repeated two and three times, with similar findings.

The alcohol curves obtained in diabetes insipidus without pituitrin therapy were similar to those observed in normal persons. There was, however, a slightly sharper rise with a slightly greater maximum level and a slightly sharper drop in these alcohol curves than in those of normal persons. Four hours after the alcohol meal the concentrations were approximately the same as normal.

With pituitrin therapy, the first half of the alcohol curve was definitely lower in diabetes insipidus than without this drug (Chart 2). In addition, pituitrin caused the alcohol concentration in the urine to rise less than that in the blood within half an hour after the alcohol meal, and to a slighter degree within one hour after it. The second half of the curve was much the same as without the administration of pituitrin.

Pituitrin appeared to prevent the usual rise in the alcohol concentration in the blood and urine after the alcohol meal. This may be due to the pituitrin's having inhibited the absorption of alcohol, or to the fact that in uncontrolled diabetes insipidus the alcohol is not metabolized so quickly as in the controlled disease.

It was noted that the blood and urine alcohol levels were higher when the alcohol was taken without pituitrin than with pituitrin. After the administration of pituitrin each specimen of urine usually amounted to only 10 or 20 cc., whereas without pituitrin the volume was as high as 400 cc. This indicates that the kidneys acted as an inert membrane and that the alcohol passed through them into the urine by simple diffusion, as suggested by Ambard,[12] Widmark[13] and others. Con-

sequently the largeness of the volume of urine did not dilute its alcohol content.

It was of unusual significance that the toxic effect of alcohol was more marked and more prolonged with pituitrin therapy, even though the blood and urine alcohol levels were lower. The toxic symptoms increased so much that pituitrin administered with the alcohol put 3 of the patients to sleep; 1 patient, however, showed no special change. All the patients seemed to experience a more immediate effect of the alcohol without pituitrin.

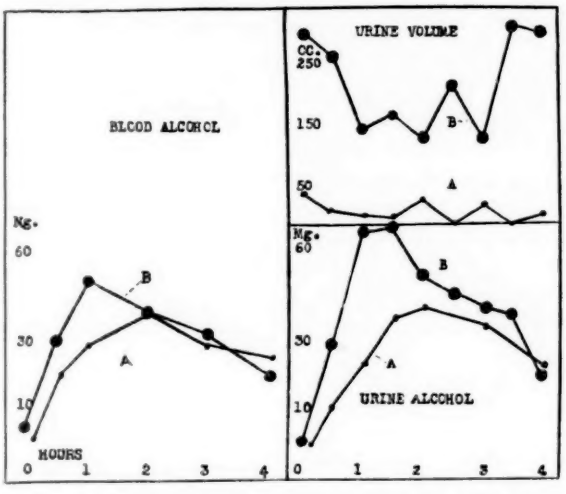

Chart 2. *Blood and Urine Findings in a Case of Diabetes Insipidus.*

Simultaneous curves showing the blood and urine alcoholic contents and the urine volume in a man with diabetes insipidus after the ingestion of 35 cc. of absolute alcohol, with (A) *and without* (B) *pituitrin therapy.*

The interpretation of these results is controversial, as is the whole problem of alcohol tolerance. However, pituitrin possibly caused the retention of a greater amount of alcohol in the tissues, giving intensified alcoholic symptoms. Under such circumstances the blood and urine alcohol might not give a proper indication of the alcohol concentration in those tissues.

FORCED WATER INTAKE AND ALCOHOL TOLERANCE

An attempt was made to produce a condition similar to diabetes insipidus in 5 normal persons by having them drink large amounts of water during the test period, so that they voided large volumes of urine. Four or five days later the tests were repeated with 1 cc. of Pitressin being given intranasally, when the usual dose of alcohol was ingested and the urine output was small. These tests were compared with those of the controls who received no Pitressin or extra water.

The increased water intake produced a slightly greater concentration of alcohol in the blood and urine during the first one and a half hours after the ingestion of alcohol than occurred during the control test (Chart 3). It was interesting that the alcohol concentrations were not diluted, although the volume of urine was increased more than tenfold at times. Miles[14] also found that changes in the amount of urine per minute did not influence significantly the alcohol concentration in the urine. In contrast, Pitressin perceptibly decreased the volume of urine and the alcohol concentration in the blood and urine at two hours after alcohol ingestion as it did in diabetes insipidus. Widmark,[15] on the other hand, found that pituitrin did not alter the blood alcohol curves in dogs.

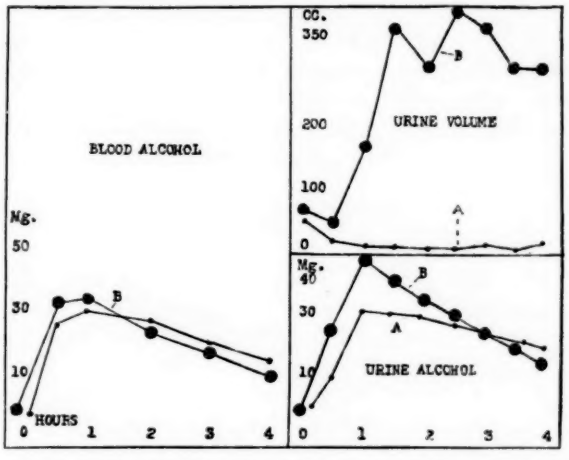

Chart 3. *Blood and Urine Findings in a Normal Man.*

Simultaneous curves showing the blood and urine alcoholic contents and the urine volume in a normal man after the ingestion of 50 cc. of absolute alcohol, with pituitrin administration (A) *on one day and the forcing of water* (B) *on another.*

The alcoholic symptoms appeared a little sooner with the forced water intake than was the case during the control tests, although the duration was about the same. In 2 persons the added water caused slightly fewer toxic symptoms and in 2 individuals slightly more. In 3 normal persons who received Pitressin administered with alcohol, the symptoms were less marked in 1 than during the control test, about the same in 1, and more pronounced in 1.

It appears from the laboratory results that forcing water might be of value in the treatment of alcoholism in increasing the elimination of alcohol, although symptomatically there was no significant change. In terms of milligrams, the amount of alcohol eliminated in the urine in cer-

tain cases after drinking alcohol and extra water was considerably increased. However, this was not of practical value since the normal individual, without forcing water, excreted in the urine 0.1 to 0.9 per cent of the ingested alcohol in four hours, compared with 1.6 to 3.4 per cent when the water intake was forced, and the amount of urine varied from 1570 to 3495 cc. Furthermore, in the marked diuresis in untreated diabetes insipidus 1.4 to 3.2 per cent of the alcohol was eliminated in the urine during the test period, compared with 0.07 to 0.22 per cent when the disease was controlled with pituitrin and the urine output was practically normal. Forcing large amounts of water only increased the excretion of alcohol in the urine to approximately 1 to 3 per cent.

ALCOHOL TOLERANCE IN DIABETES MELLITUS, WITH AND WITHOUT INSULIN THERAPY

The consideration of the immediate effect of alcohol on the blood and urine alcohol concentration and its toxicity in patients with diabetes mellitus is an important and practical problem.

The alcohol-tolerance curves were determined in 6 patients with diabetes mellitus and compared with those obtained in normal people. The blood and urine also were examined for sugar. There was no acetone or diacetic acid in the urine specimens.

There was a similar relation between the blood and urine alcohol levels in the diabetic patients without insulin therapy as in the normal individuals (Chart 1). However, the important finding was that the maximum alcohol concentration of the blood and urine about one hour after the alcohol meal was appreciably higher in the diabetic patients, although the fasting blood and urine alcohol concentrations were normal. The cause for this is conjectural. The greater rise may be due to an increased rate of absorption of alcohol. Possibly diabetic patients cannot metabolize the alcohol as rapidly as normal persons, so that there is a greater accumulation of alcohol in the blood and greater concentration in the urine. Incidentally, there was some decrease in the blood-sugar level during the test period.

Haggard and Greenberg[16] found that increasing the blood sugar in rats greatly lessened the pharmacological effect of alcohol that had been absorbed. If this were true clinically, certain diabetic patients would have an increased tolerance to alcohol. However, the toxic effect of alcohol was much the same in the diabetic patients as in the normal persons, although 2 of the former experienced considerable staggering after the alcohol meal.

There have been several reports[17–21] suggesting that insulin causes an increase in the rate of disappearance of alcohol from the blood. It seemed to us that if this were correct, it could be proved in diabetic patients in whom the blood sugar could be changed with insulin from a high to a low level in a short period of time. Consequently, the alcohol-tolerance tests were repeated in these patients after an interval of several

Table 1. *Alcohol Tolerance in Diabetes Mellitus without and with Insulin Injection.*

Case No.	Determination	Control Test — Hours after alcohol ingestion									Insulin Administered* — Hours after alcohol ingestion								
		0	½	1	1½	2	2½	3	3½	4	0	½	1	1½	2	2½	3	3½	4
		mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %	mg. %
1	Blood sugar	315	310	303		266		266		261									
	Blood alcohol	0	76	63		40		31		13									
	Urine alcohol	0.9	51	93	72	48	34	25	17	6									
2	Blood sugar	290	305	272	278	268		268		266	336	293	276		195		99		89
	Blood alcohol	0	78	—	52	44		37		20	3.0	78	69		45		27		19
	Urine alcohol	0.6	31	88	77	59	61	46	41	31	0.3	9	31	49	51	48	44	32	25
3	Blood sugar	167	170	181		140	129				237	168	158		55	57			
	Blood alcohol	1.0	47	63	55	47	43				6.5	54	62		46	37			
	Urine alcohol	0.4	13	88	83	64	52				0.8	18	91	81		59			
4	Blood sugar	110	131	126		103					186	162	102		87		72		79
	Blood alcohol	0	43	51		48					0	45	54		49		32		18
	Urine alcohol	1.6	23	48	43	56					0.8	25	52	49	55	—	46	38	31
5	Blood sugar	179	183	179		166		148		147	233	154	142		82		113		127
	Blood alcohol	0	44	48		44		30		22	4.0	47	51		44		29		17
	Urine alcohol	0.5	9	61	74	55	51	43	37	30	1.3	15	65	69	64	59	49	33	22
6	Blood sugar	147	148	143		122		107		96	176	164	148		73		87		89
	Blood alcohol	0	37	49	—	40		26		9	0	49	49		41		26		14
	Urine alcohol	0	2	75	70	51	—	34	27	17	0	7	83	63	57	49	44	36	23
Average blood alcohol		0.2	54	55		46		31		16	2.7	55	57		45		29		17
Average urine alcohol		0.7	22	76	70	56	50	37	31	21	0.6	15	65	62	57	54	46	35	25

*Twenty-five units were injected in Case 2, 20 units in Case 3 and 10 units each in Cases 4, 5 and 6.

days, when 10 to 25 units of insulin were injected subcutaneously immediately before the alcohol was ingested (Table 1).

It was observed that insulin did not alter the blood and urine alcohol curves, despite the marked drop in the blood sugar to a hypoglycemic level during the test period (Chart 4). In fact,

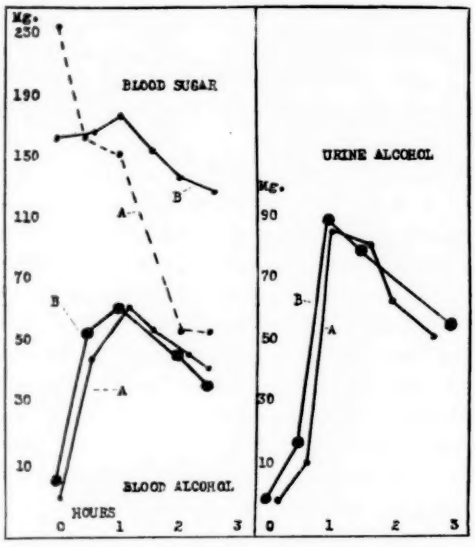

Chart 4. *Blood and Urine Findings in a Case of Diabetes Mellitus.*

Simultaneous curves showing the blood and urine alcoholic contents and the urine volume in a man with diabetes mellitus after the ingestion of 40 cc. of absolute alcohol, with (A) *and without* (B) *the injection of 20 units of insulin.*

the curves were practically identical with the control tests. It was obvious from these experiments that insulin does not affect alcohol metabolism in diabetes mellitus. Furthermore, it produced no change in the toxic symptoms caused by the alcohol. Nevertheless, during hypoglycemia a toxicity appeared which was no doubt due to the hypoglycemia and not to the alcoholemia. In addition, 0.4 to 1.0 per cent of the ingested alcohol was excreted in the urine in four hours, and this amount was not appreciably affected by the injection of insulin.

A few investigators have observed that insulin increased the rate with which alcohol disappeared from the body. Supniewski[17] showed that the subcutaneous administration of insulin into normal animals at the same time as alcohol was given caused the blood alcohol to be reduced. Aoki[18] noted a hypoalcoholemia in fowls ninety minutes after the injection of insulin. Widmark[15] found that insulin increased the rate of disappearance of alcohol in dogs as much as 200 per cent. Newman and Cutting[19] obtained a 50 per cent increase in alcohol metabolism with therapeutic doses of insulin in human subjects. Schlichting[20] also observed that insulin hastened alcohol metabolism. From experiments made on subjects with normal blood sugar, Bickel[21] concluded that insulin was effective in accelerating the speed of alcohol metabolism by lowering the blood sugar, and that when this was prevented by ingestion of sugar no increase in rate occurred.

In contrast, there is good evidence that insulin has no effect on alcohol metabolism. Hirschfelder and Maxwell[22] determined that insulin did not increase the oxidation of alcohol in the body or antagonize its toxic effect. Dell'Acqua,[23] as well as Lang and von Schlick,[24] could perceive no effect of insulin on alcoholemia. Fleming and Reynolds[25] found that the injection of insulin into human beings did not modify the concentration of alcohol in the blood after the intravenous administration of alcohol. Siegmund and Flohr[26] obtained no evidence in 3 healthy men that medication with insulin reduced the symptoms or hastened the disappearance of the signs of intoxication. These findings agree with my results obtained in the patients with diabetes.

FOOD AND ALCOHOL TOLERANCE

It has long been known that food in the stomach has an inhibitory effect on the symptoms of alcohol intoxication. This has been attributed to the influence of food in decreasing the absorption of alcohol from the stomach into the blood stream.

This problem was studied in 4 patients with diabetes mellitus, in addition to the experiments on the effect of alcohol with and without the administration of insulin. In these tests the patients took a normal-sized breakfast. Approximately one hour later they received the standard dose of alcohol. In 2 cases insulin was injected just before the alcohol intake. The results were very striking (Chart 5). It is clear that the maximum concentrations of alcohol in the blood and urine after the ingestion of food and alcohol were much lower than they were when food was omitted. The addition of insulin injection to food made no great difference in the results.

An important observation was that the alcoholic symptoms following the ingestion of food were greatly decreased and hardly noticeable. Incidentally, similar results were obtained in patients with diabetes insipidus.

Just how food taken with alcohol causes a lowering of blood and urine alcohol levels is a controversial question. The action may be explained by a decreased rate of absorption of alcohol, as suggested by Mellanby.[27] If this were

the case, the alcohol would disappear more slowly and remain in the body longer when food was taken. However, Carpenter and Lee[28] found that this was not the case. They examined the alcohol in expired air after the ingestion of alcohol and glucose, and found that it disappeared more rapidly than when no glucose was given. This raises the question whether the disappearance of alcohol is due to its combustion or to its transformation into some other substance. Southgate[29] ascribed this phenomenon not to delayed absorption but to the fact that a considerable fraction of the ingested alcohol is never manifested in the blood. This indicates that it is due to some chemical reaction. On the other hand, the experiments of Fleming

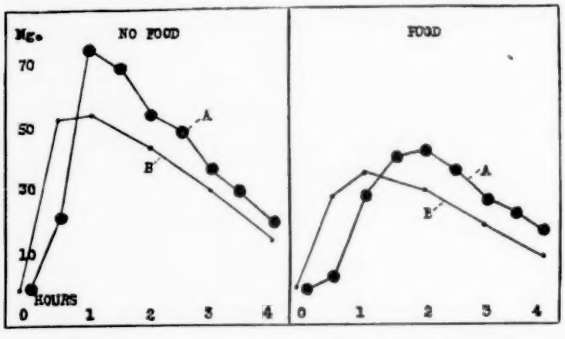

Chart 5. *Response to Ingestion of Alcohol in Relation to Food.*

Average curves for urine (A) *and blood* (B) *alcoholic contents obtained in 4 patients with diabetes mellitus after the ingestion of 0.6 cc. of absolute alcohol per kilogram of body weight, with and without the intake of food.*

and Reynolds[25] suggested that certain foods do not affect alcohol metabolism: they gave egg albumin, milk, cream and olive oil after the intravenous injection of alcohol and found no significant change in the rate of disappearance of alcohol from the blood.

DISCUSSION

The study of alcohol tolerance is a fascinating one, although somewhat perplexing. The proper explanation of the results obtained in this investigation is at present uncertain because our knowledge of the intermediary metabolism and certain aspects of the utilization of alcohol in the body is incomplete. Insulin and pituitrin have been shown to have antagonistic effects on carbohydrate[30] and fat metabolism.[31] The data here reported suggest that the two substances do not influence alcohol metabolism in opposite ways. Pituitrin decreased the blood and urine alcohol levels in patients with diabetes insipidus and in normal individuals, but in spite of this the symptoms of alcoholic intoxication were increased in the former. This suggests that pituitrin causes greater retention of alcohol in the tissues of these patients than is indicated by the concentrations of blood and urine alcohol.

The appearance of an increase in the blood and urine alcohol levels following the ingestion of alcohol in patients with diabetes mellitus indicates that there is an increased rate of absorption of the drug in this disease, or that the alcohol cannot be metabolized as quickly as in normal cases. Even insulin had no effect on the blood and urine alcohol levels or on the symptoms of alcohol intoxication in diabetes mellitus.

No evidence was supplied that alcohol is more harmful to the diabetic patient than to the normal person, since the toxic effect of alcohol was much the same in both, although the alcohol curves were appreciably higher in the former. However, alcohol should not be recommended for the diabetic patient, because an alcoholic odor in his breath may lead to confusion in the diagnosis of coma due to insulin or acidosis.

It was interesting that food caused a decrease in the concentrations of blood and urine alcohol and also in the toxic symptoms following the ingestion of alcohol. Although this has been attributed to a physical action whereby the rate of alcohol absorption is decreased, there is other evidence which suggests that the cause may be a chemical reaction in which the metabolism of alcohol is increased.

The general relation between the concentrations of blood and urine alcohol appears to be that during the period of alcohol absorption its concentration in the blood is higher than that in the urine. One hour after the ingestion of alcohol the alcohol level of the urine was slightly higher than that of the blood and remained so during the period of alcohol decline. Nevertheless, the two levels were very close, even though large amounts of urine were voided. This suggests that alcohol passes through the kidneys into the urine by a simple process of diffusion. The reason for this slight difference has been explained[32] by the alcohol level's being slightly higher in arterial than in venous blood, and consequently giving the slight variation between the alcohol concentrations of the urine and of venous blood.

SUMMARY

This paper presents a study on some alcohol tolerance tests in normal persons and in patients with diabetes insipidus and diabetes mellitus following the ingestion of 0.6 cc. of absolute alcohol per kilogram of body weight. The influences of insulin,

pituitrin, food and forced water intake on these tests were determined.

Symptoms of alcoholic intoxication appeared in these people when the blood and urine alcohol concentrations were below 63 mg. per cent, in comparison to much higher levels reported by others.

The blood and urine alcohol curves after the test meal were very slightly higher in patients with diabetes insipidus than in normal persons. Pituitrin reduced the blood and urine alcohol levels in both these groups; yet notwithstanding this it increased considerably the symptoms of alcoholic intoxication in the former.

In the patients with diabetes mellitus the blood and urine alcohol levels after the ingestion of alcohol were appreciably higher than normal, although the symptoms of alcoholic intoxication were much the same. The administration of insulin had no effect on the alcohol curves or on the symptoms of alcoholic intoxication. It appears that alcohol is no more harmful to the diabetic patient than to the normal individual, although an alcoholic odor on the breath may cause confusion in the diagnosis of coma due to insulin or acidosis.

The ingestion of food before the ingestion of alcohol produced a striking decrease in the blood and urine alcohol levels and in the symptoms of alcohol intoxication in diabetic patients.

The diuresis resulting from forced water intake and uncontrolled diabetes insipidus did not dilute the concentration of alcohol in the urine, but increased the total amount of alcohol excreted therein. Nevertheless, forcing water in the case of an alcoholic patient would not produce any appreciable results in therapy, because the total alcohol eliminated by this method is small compared to the amount of alcohol ingested.

189 Bay State Road.

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REPORT ON MEDICAL PROGRESS

ABDOMINAL SURGERY

ARTHUR W. ALLEN, M.D.*

BOSTON

RECENT advances in surgery have been attained through improvements in technical procedures, more perfectly controlled anesthesia and a gradually increasing knowledge of pathologic physiology.

PREOPERATIVE AND POSTOPERATIVE CARE

We have often seen a well-accomplished surgical operation fail because of improper preparation of the patient and a lack of understanding of the physiological and biochemical factors at fault. Valuable contributions have been made in this field within recent years. Since in such a large proportion of cases the principles involved apply to patients with intra-abdominal lesions, we shall stress their importance in this paper.

It is incredible but true that many surgeons have no hesitation in subjecting patients to a serious abdominal operation in a state of fatigue and general debility, with little or no thought of the effect of the superimposed trauma. Thus, a tired business man or a housewife, having redoubled his or her efforts for days and weeks, enters the hospital late in the day and is subjected to a major operation early the next morning. In casting about for the reasons why such a patient has done poorly after operation, the last thought is usually the obvious one: a tired horse has been entered in a race. This mistake, which is made even by men of high intelligence, is one of the chief causes of disaster. A few days of training in the hospital environment can often be utilized to good advantage. A correction of water and salt balance, a preliminary transfusion, and an evaluation of sedatives in addition to rest and nourishment often make the difference between a smooth and a stormy convalescence, and even between life and death.

In the postoperative care of this patient one must have a baseline from which to start, so that the patient's needs can be met in an orderly manner and not by general rule. Fluid must be administered, but the amount may be too great in a given period for the patient in question, producing a fatal pulmonary edema or cardiac dilatation. Salt must be limited to his physiological needs or water will be retained within the tissues. Glucose can be utilized up to certain limits, and a moderate excess is offset by loss through the kidneys. A reasonably safe rule is: use 50 gm. of glucose in each liter of normal saline solution and of distilled water, alternately. A patient with a high intestinal fistula or an inlying Levine tube needs more salt than one whose hydrochloric acid is retained within the intestinal tract. The average adult patient needs approximately 3 liters of fluid, evenly distributed in each twenty-four hours. A rough guide is the urinary output, which should average not less than 1000 cc. per day. Depleted patients and those who have had large resections should be routinely transfused; those who have lost an abnormal amount of blood should receive enough to bring the blood content up to a low normal level.

Patients should receive a proper amount of sedatives, if any is to be given; where there is need for maintaining the tone of the small bowel, morphine is the drug of choice. The only guide as to the amount should be the rate of respiration, which should not descend below 12 per minute. Pitressin has been advocated by some observers; its action is primarily upon the large bowel; if used, its administration should be started on the operating table and continued until the need for it has passed, that is for forty-eight or seventy-two hours. If it is given after distention has taken place, a rupture of the large intestine may follow. One must realize that after laparotomy there is a stage of physiologic ileus. Food should be withheld and the tone of the small bowel maintained until normal peristalsis has returned. If this precaution is neglected the patient's abdomen will become distended and vomiting will ensue. Fine and Levenson[1] have contributed much to the problem of gaseous distention after operation. They have shown that milk, orange juice, carbohydrates and protein in the order named, all produce gas. Small amounts of water, followed by consommé, tea, cooked cereal, milk and lime water should be used until intestinal elimination has become adequate. Postoperatively patients should be exercised by deep breathing, frequent turning, and movements of the arms and legs. The restless patient almost never develops phlebitis and pulmonary embolus, while the fat, lethargic individual is prone to do so.

*Lecturer in surgery, Harvard Medical School; chief of the East Surgical Service, Massachusetts General Hospital.

GASTROSCOPY AND PERITONEOSCOPY

The most useful new adjuncts to diagnosis aside from laboratory tests are the gastroscope and the peritoneoscope.[2] Lesions within the stomach are often so baffling to the roentgenologist that he welcomes the aid of the gastroscopist. Experience in this field will often lead to a correct interpretation of the pathologic process or lack of it, and thus bring about the logical therapeutic approach. Thus early manifestations of malignancy may be recognized in a curable state, and benign lesions may be treated on a conservative basis with greater assurance of success.

Peritoneoscopy is a comparatively safe and simple procedure. The patient has little discomfort during it, and may be up and about within twenty-four hours. When the usual methods of diagnosis fail to solve an intra-abdominal problem, we can often settle the question by peritoneoscopy. So far, one of its most useful applications has been in advanced carcinoma of the stomach. If there are metastases to the liver or peritoneum exploratory laparotomy should be avoided, since under these circumstances cure is impossible and palliative surgery is rarely worthwhile; also, one third of the patients in this group succumb during their postoperative convalescence. In a high percentage of doubtful cases of cancer of the stomach, Benedict[2] has been able to determine the operability in a correct manner, an accomplishment which has reduced to a minimum the number of useless exploratory laparotomies in this disease. Peritoneoscopy is helpful in many other doubtful intra-abdominal conditions, and more use will be made of it as experience grows and results are properly evaluated.

HEALING OF WOUNDS

Methods of incision have changed little in many years, and aside from the careful avoidance of nerve injury a surgeon may safely employ any approach to a given area that best suits his purpose. Considerable attention has been given to the use of suture material in connection with the healing of wounds, and it seems that surgery throughout the country is progressing toward those ideal principles so carefully laid down by Halsted more than a quarter of a century ago. Cleanliness, gentleness, sharp dissection, hemostasis, avoidance of dead space and the prevention of foreign bodies in the wound have been accepted as the criteria of good surgery and perfect wound-healing. Fine silk, instead of the usual sizes of catgut, causes less foreign-body reaction in the tissues and hence results in better wounds. Fine, malleable steel wire causes even less reaction than silk, and is favored by some surgeons. In certain types of wounds, particularly where contamination is unavoidable, there is a tendency to employ no sutures except properly placed, through-and-through strands of pure silver wire, or especially treated non-porous, heavy, braided silk, thus avoiding all possibility of foreign material within the wound. Dehiscence, wound infection and hernia in the scar may thus be reduced to a minimum.

HERNIA

Roscoe Graham's[3] explanation of the mechanism of sliding hernia is of the utmost importance. Sliding hernia of the sigmoid has been particularly difficult to reduce and repair through an inguinal incision alone. The bowel herniates between the leaves of its mesentery, and if the abdominal cavity is opened above through a separate incision and traction is exerted on the sigmoid, the bowel is easily reduced; the two leaves of the mesentery come together in a normal fashion. The defect in the inguinal canal is best repaired through the usual oblique incision after the abdominal incision has been closed.

Williams[4] advocates a more frequent abdominal approach in the repair of inguinal hernia. This is especially advantageous when dealing with undescended testicle and hernia.

Payne[5] and others favor the use of strips of fascia from the external oblique muscle in the repair of femoral hernia. Such strips have been used ever since McArthur suggested them in the repair of the defects of inguinal hernia.

Gallie and Le Mesurier's[6] contribution — the repair of the hernia with strips of fascia lata — has been widely adopted, and with great success. Certainly many defects could not otherwise be corrected.

APPENDICITIS

The educational campaign against the use of cathartics in acute abdominal discomfort has succeeded in reducing the mortality from appendicitis in Philadelphia.[7] A program of widespread activity in this direction elsewhere throughout the country is in order.

In adult patients with peritonitis complicating appendicitis, there is considerable evidence in favor of delayed surgery. The highest mortality occurs in such patients when operated on between the third and sixth days of the disease. This explains the increasing mortality in acute appendicitis. Many cases are still treated expectantly in the home, even under the guidance of a physician. As soon as rupture has taken place, with the resultant spreading peritonitis, the patient is sent to the hospital; if operation is immediately undertaken,

death is more likely than at any other stage of the disease. Conservative physicians in previous decades continued their expectant type of treatment at home until there was spontaneous cure, or death occurred, or an obvious localized abscess developed. Under this regime the mortality was lower than it is at present. Thus it has become apparent that there is a stage of spreading peritonitis which is better treated by supportive measures until localization has taken place. Children do not seem to tolerate this method of treatment as well as do adults.

The general attitude on drainage of the abdominal cavity in appendicitis has changed decidedly. There was a time when the dictum was, "In case of doubt, drain"; it is now definitely the reverse. Except in cases with the formation of a localized abscess, drainage is contraindicated. The excess fluid may be aspirated with care and gentleness with the suction tip, and the peritoneum sutured tightly. The contaminated layers of the abdominal wall are often drained to advantage.

The detailed modern adaptation of Ochsner's regime has become of great importance in the successful management of peritoneal involvement, both as a preventive and as a curative procedure.

BILIARY SYSTEM

The tendency of patients with obstructive jaundice to bleed can now be controlled by the use of vitamin K and cholecystic acid. Careful laboratory studies by Stewart[8] and others indicate that the dosage of this vitamin and the period of time necessary to prepare such patients for operation will soon be standardized. So far, this substance cannot be obtained commercially, but it will be forthcoming at an early date. The laboratory test for the prothrombin level of the blood will soon be a matter of routine. Thus one of the chief causes of death in obstructive jaundice will be eliminated.

Much evidence has been submitted in favor of early operation in acute gall-bladder disease. It appears that the mortality in cases so treated is lower than in similar groups in which the surgeon postpones operation in the hope that the disease will quiet down. These cases are not emergency ones, to be operated on at night with inadequate assistance, but should be evaluated after dehydration has been combated. If greatly improved in twelve hours as indicated by less fever, a decrease in leukocytosis and local tenderness, one may safely wait longer; in the majority of cases the patient will come to operation a week or ten days after the acute onset in a better condition for operation. The gangrenous gall bladders that need early surgery do not quiet down, but the high white-cell count, fever and local tenderness persist after the loss of fluid has been offset. In these cases operation should be carried out as soon as reasonably ideal conditions can be obtained.

In cases of chronic gall-bladder disease, artificial edema greatly facilitates the preservation of an adequate amount of serous coat for peritonization of the hepatic fissure. This was brought to my attention by Dr. Alfred M. Rowley,[9] of Hartford, Connecticut, and was probably first suggested by Bevan.[10] Salt solution injected through a fine needle beneath the serous covering of the gall bladder makes its enucleation much easier—whether this is done from the fundus toward the ducts or from below upward. We have all become conscious of the importance of the common duct, and in diseases of the biliary tract realize that injury to this structure must be avoided at all costs. Anomalies of the ducts and the blood supply are found more frequently than has been realized. A Russian anatomist reports 10 per cent of anomalies in a large number of dissections. There are fewer injuries to the common duct if the gall bladder is carefully and bloodlessly dissected out from the fundus toward the ducts. One disadvantage of this method is the possibility of forcing small stones through the cystic duct into the common duct, but this can often be prevented by placing a clamp on the cystic duct before beginning the dissection. The common duct should be explored and its outlet into the duodenum gently and gradually dilated with suitable bougies, if necessary. The width of dilatation of the sphincter of Oddi should always be less than the diameter of the duct, and never more than the size of the stones that might be hidden within the hepatic ducts. A 7-mm. Bake's dilator or a No. 21 French bougie is usually adequate. Indications for exploration of the duct are numerous. Frequent attacks of pain, vomiting, chills and fever, jaundice, small stones, a large cystic duct, a large or thickened common duct, thickening in the head of the pancreas, palpable stones within the ducts and a stoneless gall bladder are some of the commoner reasons for exploration. Over one third of the gall-bladder operations at the Massachusetts General Hospital are accompanied by duct exploration, and in approximately one third of these, stones are removed.

PANCREAS

In acute pancreatitis there is a tendency toward a nonoperative, supportive type of treatment. The diagnosis is often difficult, but can finally be determined by paracentesis with a small needle. If thin, bloody fluid is obtained in the presence of other adequate symptoms and signs, the diagnosis should be considered as established. Operation

may be undertaken on an erroneous diagnosis, but few cases should be treated conservatively without the confirmation of paracentesis. Indications for surgery are unrelievable pain and nausea, jaundice, and a palpable mass. Biliary decompression relieves the pain; jejunostomy for feeding and a Levine tube in the stomach relieve the nausea. Drainage of the pancreas itself should be limited to adequate drainage of the lesser peritoneal cavity.

Adenomas of the pancreas are being reported from time to time. These cases are apt to be suspected by the psychiatrist, since the convulsive phase of hyperinsulinism is prone to be interpreted as a form of epilepsy. Marked emaciation and a prolonged false psychosis lead to the wrong interpretation of symptoms. The results in a successful adenectomy are dramatic. The adenomas are often small, and may be situated in the head of the pancreas and on the posterior side. They have a characteristic cherry-red appearance and are usually single. Attempts at subtotal pancreatectomy in the hope of including a small, non-palpable adenoma are apt to be disappointing, since the tumor may well be left in the remaining portion of the organ.

Carcinoma of the pancreas, when limited to a small area near the papilla of Vater, may be brought to light by jaundice while still resectable. Whipple, Parsons and Mullins[11] have developed a rational two-stage attack to relieve this situation, and have subjected 8 patients to this procedure; 1 of them lived three years after operation before recurrence in the liver caused death. In this operation the gall bladder is anastomosed to the gastrointestinal tract and the common duct is ligated. At a second operation two weeks later the duodenum and the head of the pancreas are removed en bloc. The duct of the pancreas is carefully ligated; the cut edge of the pancreas is sutured and drainage to this area is established by cigarette wicks. Continuity between the stomach and intestine is established either at the first or the second stage.

Stones in the pancreas are of the same consistence as salivary calculi, and are easily seen in an adequate roentgenogram of the region. Haggard[12] has collected about 140 cases from the literature, and some from his own practice. He concludes that symptoms of continued pain radiating to the back with nausea and vomiting warrant surgical interference. Following operation, fistulas are prone to develop if the stones were in the main duct. Drainage may continue for several years but finally ceases.

STOMACH

Ulcers of the stomach should be considered malignant until proved benign. Under ideal treatment the ulcer should heal entirely within six weeks. Ulcers with cancer in them may improve under medical management, as shown by an apparently smaller crater in the roentgenogram and loss of symptoms. This may result in too long an interval between examinations, and the passing by of the stage wherein the lesion is curable by adequate resection. Recurrence of ulceration and profuse bleeding is also an indication for surgery. If the lesion is unquestionably benign, the ulcer may be destroyed by excision or cauterization, combined with some procedure aimed at increasing the alkalinity of the stomach contents. Jejunal ulcers do not occur following gastroenterostomy for gastric ulcer. Polyposis of the stomach, leiomyomas and suspicious ulcerations are best treated by gastrectomy. Polyps bleed and degenerate into malignancy; leiomyomas bleed periodically and spread locally. Questionable ulcerations are often found to be malignant.

Pre-pyloric ulcerations are nearly all malignant; those on the lesser curvature are so in approximately one third the cases.

Carcinoma of the stomach offers an operability of approximately 30 per cent, including the patients subjected to total gastrectomy. Approximately 20 per cent of those surviving subtotal gastrectomy live five years or more after operation.

Twenty-four total gastrectomies have now been done at the Massachusetts General Hospital; in many of the cases the prognosis was unfavorable from the beginning. The operative mortality is still 50 per cent, owing to the inclusion of cases with pancreatic and colonic involvement. The survivors had a more comfortable life, varying from a few months to four and a half years. Those dying of liver recurrence had a more comfortable exitus than did those beyond the reach of resection who died of starvation. Jejunoesophagostomy, enteroenterostomy between the loops of the jejunum and jejunostomy for feeding formed the best combination of procedures. The loop of the jejunum is attached to the diaphragm by a row of sutures running entirely around the anastomosis between the end of the esophagus and the side of the jejunum.

DUODENUM

Ulcer of the duodenum is a medical problem. The complications warrant surgical interference.

In the first place, acute perforation must be closed; the simplest method is that described by

Roscoe Graham,[13] which consists of holding a graft from the omentum over the opening by means of sutures passed above, below and through the ulcer and tied loosely over the graft.

Secondly, cicatricial obstruction may take place when the ulcer is near the pylorus and the condition has had many flares and cures. This is a mechanical problem occurring mainly in patients beyond middle life; the condition responds well to simple gastroenterostomy or pyloroplasty.

Thirdly, profuse bleeding is usually associated with ulcers of the posterior wall which have eroded the pancreaticoduodenal artery. Spontaneous remission usually occurs in patients under fifty years of age. If they are nearing this age, a radical attempt to rid them of the tendency to ulcer should be made in a quiescent stage — usually not under six weeks from a given period of severe bleeding. The tendency for ulcers to recur and to become intractable to conservative measures is due to their deep penetration into the pancreas and the complete loss of continuity of the duodenum in this area. Patients over fifty years of age with this lesion have enough rigidity in the wall of the bleeding vessel to interfere with the organization of an adequate clot, and death from hemorrhage occurs in one third of these cases. Radical operation during the phase of bleeding is rarely indicated, but when it is, the indication is definite, and the operation if undertaken within forty-eight hours of the onset of hemorrhage carries little more risk than does an operation by election.

Fourthly, intractable symptoms occur in about 5 per cent of duodenal ulcerations. These may be due to economic or social circumstances. Radical subtotal gastrectomy is indicated in such cases. This should include the excision of all the gastric mucosa from the pylorus to the junction of the middle and upper thirds of the stomach, and of the first portion of the duodenum when practical.

Fifthly, gastrojejunal ulcers rarely respond well to conservative measures. If bleeding and pain persist and roentgenograms show no progress, surgery should be undertaken. When feasible a subtotal gastrectomy should be done. If operation is postponed a gastrojejunocolic fistula may develop, and surgery then becomes imperative and increasingly dangerous. The fistula should be excised in an aseptic manner, with the restoration of normal function. After recovery from the operation, reactivation of the original duodenal ulcer is liable to occur; then a subtotal gastrectomy in a clean field gives maximum protection.

REGIONAL ENTERITIS

Regional enteritis has become a recognized entity. Long classified as non-specific granuloma and often diagnosed clinically as tuberculosis, it follows a pattern sufficiently definite to warrant considerable interest. The symptoms are vague and somewhat variable: abdominal pain with diarrhea, loss of weight and tenderness in the right lower quadrant are often followed by the formation of an abscess, with ultimate fistulous tracts running to the perineum or to a previous exploratory or appendectomy incision. The commonest site is the terminal ileum, but the cecum is often involved. Other segments of small intestine are affected at times, and more rarely segments of the colon become diseased. Reichert and Mathes[14] have reproduced a similar condition in animals by injecting the lymphatics of the ileocecal region. Crohn, Ginzburg and Oppenheimer[15] have done much to clarify the pathologic picture and have added greatly to the knowledge of the disease. The etiology remains unknown. The most successful form of treatment thus far employed is a wide, radical resection of the diseased bowel with its node-bearing areas. Mixter[16] has reported a series of approximately 20 cases so treated, with excellent results. Recurrence occurs in a certain proportion of cases; it may be due to inadequate removal of the lymphatics, or to the inclusion in the resected portion of an insufficient margin of apparently normal bowel.

COLON

Idiopathic ulcerative colitis warrants permanent ileostomy in approximately one third of the cases, and according to McKittrick and Miller[17] about half these patients require total colectomy before they can lead reasonably normal lives. Patients with this condition should not be allowed to become too ill or depleted before one resorts to surgery, or the mortality will be unjustifiably high.

Polyposis involving all or various segments of the colon justifies colectomy. There is a marked familial tendency in this disease. The rectum can be saved in many cases, and the polyps in this segment can be watched and fulgurated at intervals through the proctoscope.

Carcinoma of the colon is a frequent condition, and such a lesion should be suspected if there is any change in bowel habit, blood or mucus in the stools, anemia associated with disturbances of the digestive tract, or a palpable intra-abdominal mass; loss of weight and obstructive symptoms indicate an advanced lesion, often inoperable. The total operability, however, is high, — over 70 per cent, — as is the curability — over 50 per cent. The operative mortality averages 15 per cent.

The right colon should be resected in two stages. A preliminary ileotransverse colostomy should be followed ten or fourteen days later by a

colectomy on the right. The supposition of protection of the peritoneum by a previous operation is substantiated by research in this field by Coller, Ransom and Rife.[18] Lesions in the transverse colon should be attacked by a preliminary proximal drainage. The left colon may be decompressed by cecostomy, and this has been a very satisfactory procedure preliminary to resection. With the use of a large tube the colon can be adequately prepared, and by infolding the cecum on the tube at the time of cecostomy, it can be made to heal spontaneously when no longer needed. Devine[19] has recently described a method of "defunctioning" the diseased left colon and rectum by a preliminary double-barreled proximal transverse colostomy. The bowel is divided between clamps with a cautery, and the limbs of the bowel are sutured together over a distance of 8 cm., the ends being left long enough to be brought to the surface and separated by the entire abdominal wall. The distal segment can be irrigated so that in two or three weeks it is actually free of infection. The diseased bowel is then resected through a clean field. When the need of the proximal colostomy has passed, the spur is crushed by special clamps, the blades of which are applied separately and then brought into contact from the surface. This method has much to commend it and is likely to be extensively used, especially by those who have not perfected aseptic anastomosis and those who have been disappointed in the adequate functioning of cecostomy.

Modifications of the Parker – Kerr type of aseptic intestinal anastomosis have given the lowest mortality of any method used at the Massachusetts General Hospital. Accurate attention to details in the technic is essential, and if properly executed these procedures will yield excellent results.

CARCINOMA OF THE RECTUM

Radical combined abdominoperineal resection after the method of Miles[20] is the procedure of choice. It can be safely applied to approximately 75 per cent of cases. Careful preoperative preparation, attention to technical details, spinal anesthesia and supportive postoperative measures have reduced the mortality to approximately 10 per cent. Patients who are poor risks, those with large infiltrating lesions and those with any degree of obstruction should be operated on in two stages. The preliminary stage may be any of the proximal drainage procedures that are found effective in the hands of the operator. Devine's[19] defunctioning transverse colostomy more nearly meets all requirements than any other so far advocated.

INTESTINAL OBSTRUCTION

The application of continuous suction to an inlying stomach catheter, as employed by Wangensteen and Paine,[21] has eliminated the need of many operations for ileus. If the obstruction is complete and of a mechanical nature, this procedure may be carried on to a period when operative interference is impossible. In maintaining the body fluids and salts and supporting the patient by blood transfusions, one may not realize that obstruction exists. Thus if the total fluids withdrawn from the upper gastrointestinal tract are closely watched, and a time limit of not more than three days of such treatment is set, the opportunity for surgical intervention will not be lost.

Miller and Abbott[22] have devised an ingenious long triple tube of nasal-catheter size that can be introduced slowly and, by the aid of a small balloon near the tip, passed all the way down through the intestine. That this tube should pass down the intestine and at the same time allow suction of fluid contents seems amazing. That it will in certain cases reach the actual point of obstruction, and in cases where the obstruction is not complete will relieve the patient and avoid a dangerous operation, is an established fact. Practice is necessary to get the end of the tube through the pylorus, and frequent roentgenograms are needed until this has been accomplished. Dr. George W. Holmes[23] has made the helpful suggestion that iced drinks will cause the pylorus to open and this point may be of great aid in the successful use of the tube. Scudder, Zwemer and Truszkowski[24] have studied a large series of cases of intestinal obstruction. Many of the symptoms in these patients, they found, are due to a heightened blood-potassium level. This may be produced in other ways, such as fistula of the small bowel, adrenal insufficiency and potassium poisoning. Lethal levels of blood potassium are frequently found as a result of experimental obstruction, and occasionally in patients. Rapid return to a normal level follows operative removal of the obstruction.

MECKEL'S DIVERTICULUM

In adults, this condition is rarely diagnosed correctly before operation. In the acute stage of inflammation the preoperative diagnosis is usually intestinal obstruction. In children the diagnosis is frequently made on the basis of abdominal pain and blood in the stools. Owing to embryonal rests of gastric mucosa within the diverticulum, all the symptoms and signs of so-called peptic ulcer may be present — even, on occasion, those of acute perforation. One of the most

reliable diagnostic points is tenderness directly beneath the umbilicus.

CALCIFIED MESENTERIC LYMPH NODES

This condition is so frequently seen in patients without abdominal symptoms that inadequate significance has been attached to it. Calcification is probably the result of healing in certain cases of mesenteric adenitis. Approximately 80 per cent of children having calcified nodes on routine examination have no history of any prolonged abdominal symptoms. In the remaining 20 per cent, half of them become symptom-free in a period varying from a few months to a few years on a conservative dietary and hygienic regime. In a small number of cases symptoms persist into adult life. The symptoms are those of vague abdominal discomfort that may occur daily without actual interference with routine life or may cause recurring bouts of acute abdominal pain with nausea and vomiting. Appendectomy is often done during or following one of these acute episodes. The attacks may recur with such frequency and so little attention be paid to them by the physician that the patient is labeled a neurasthenic or hypochondriac. Careful evaluation is justifiable and, in certain cases, excision of the calcified masses is followed by a complete relief of symptoms and a restoration to robust health of an individual previously handicapped by chronic invalidism. The tenderness usually starts just at the right of and slightly below the umbilicus, and extends toward the left upper quadrant, following closely the mesenteric vessels. In a group of such adult and adolescent patients studied by Allen and Howe[25] at the Massachusetts General Hospital, it was found that appendectomy alone relieved 50 per cent. Appendectomy and excision of the chief mass of calcified nodes relieved 95 per cent. Those unrelieved were found to have such an extensive distribution that removal of the nodes was impracticable.

264 Beacon Street.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 25071

PRESENTATION OF CASE

A twenty-year-old, single Russian Jewess was admitted complaining of pain and swelling just below the right knee.

About five months prior to admission the patient first noticed that while climbing stairs she had pain in the right leg just below the knee. About one month later she noted the appearance of a swelling over the painful area. The swelling was tender, the tenderness being most marked along the upper anterior surface of the left tibia. This swelling gradually increased. At times it was hot and red. She noted most discomfort in climbing stairs, running, or bending over.

In the past she had been in good health except for diphtheria two years before entry. Her tonsils had been removed about two years before admission. The family history revealed no evidence of familial disease.

Physical examination showed a well-developed and nourished woman in no acute distress. Examination of the head, neck, chest and abdomen was negative. The blood pressure was 118 systolic, 72 diastolic. On the right leg, just below the tibial tubercle, there was a hard, bony swelling about 2 cm. in diameter. It was tender to pressure. The skin was not red or edematous. There was no fluctuation. All motion of the knee was normal.

The temperature was 97.8°F., the pulse 72, and respirations 17.

Examination of the urine was negative. The blood showed a red-cell count of 5,000,000 with 85 per cent hemoglobin, and a white-cell count of 8600 with 72 per cent polymorphonuclears. A blood Hinton test was negative.

X-rays showed a cyst-like area of decreased density, about 3 cm. in diameter, 5 cm. below the proximal epiphyseal line of the right tibia. The lesion was anterior and medial, bulging above the surface of the remainder of the bone. The cortex was markedly thinned in this area. There was increased density of the bone surrounding the lesion. No changes were visible in the soft tissues.

The patient's chart remained normal. On the fourth hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. CHANNING C. SIMMONS: There are a few data that I should have liked to have but which are not available: first, x-rays of the other bones; secondly, the blood-chemistry findings; thirdly, a more detailed history; and, finally, a more careful physical examination, although presumably it was negative.

This is the x-ray film of the lesion. It is evident that this patient had a lesion in the upper end of the tibia situated about 5 cm. below the knee joint. In all cases of bone tumor three chief conditions should be considered. First, is it a metabolic or generalized bone disease, such as osteitis fibrosa or Paget's disease? Secondly, is it an inflammatory condition, such as tuberculosis, syphilis or osteomyelitis? Thirdly, is it tumor, either primary or metastatic?

Let us take up the first group — the metabolic diseases. We have nothing that points to such a condition, although I should like information on several blood constituents and more data on the differential count. We also should have x-rays of the other bones, to rule out the possibility of generalized disease. If the tests were all negative, and we may assume they were, I think we can exclude the various metabolic diseases. The patient is young for Paget's disease.

The chief inflammatory conditions to be considered are tuberculosis, syphilis, which is always a possibility, and osteomyelitis. The Hinton test was negative; this presumably rules out syphilis, although in bone syphilis serological tests are negative in 35 or 40 per cent of the cases. There is nothing else that points toward syphilis. I am sorry to say, I am not very conversant with the radiological appearance of tuberculosis, but to me the film does not suggest it. I should expect more change in the joint, and more physical disability than she had. Osteomyelitis may be anything from the acute form to the chronic form known as Brodie's abscess. With that condition we usually have a history of respiratory infection such as tonsillitis, nasal infection or pneumonia preceding the attack of bone pain. She gives no history of such. In Brodie's abscess the x-ray film shows a bony defect with thickening of the bone about it, such as this film shows, so such a diagnosis has to be seriously considered. Usually, however, there is some systemic reaction. The white count should be 10,000 or 12,000 rather than 8000, and the temperature slightly elevated rather than 97.8°F. I should rule out tuberculosis and

syphilis and leave osteomyelitis to be considered later.

There is no evidence in the physical examination of a primary tumor of which this might be a metastasis. Of the primary tumors of bone one would have to consider the nonmalignant and the malignant tumors. The nonmalignant types include osteoma, solitary bone cyst, which one may call tumor, and osteochondroma, which might occur in that region, though most chondromas give less the effect of thickening of the bone about the lesion. Another form of tumor is the subperiosteal cyst found in cases of neurofibromatosis; this patient had no areas of pigmentation on her body and no subcutaneous neurofibromas.

Of the malignant tumors to be considered the first is osteogenic sarcoma which usually shows bone destruction and bone formation; but there is no reactive triangle such as one usually sees. Ewing's sarcoma rarely arises in this location and usually shows more bone destruction, although atypical radiographs are not uncommon. It might be an osteogenic sarcoma but I doubt it.

My opinion is that this is either a Brodie's abscess or a chondromatous tumor with a superimposed inflamed bursa. I think a personal examination of the case would have made the diagnosis easier.

Dr. Tracy B. Mallory: Have you any comment, Dr. Hampton?

Dr. Aubrey O. Hampton: The only thing I can add is that if we had films to show the regional soft tissue over that lesion we should see subcutaneous edema above and below, which would probably rule out tumor and make it more likely to be inflammatory. Furthermore, the dense margin around the bony defect ought to be thinner if it were due to chondroma. I should agree with Dr. Simmons that the lesion is probably a Brodie's abscess.

Clinical Diagnosis

Brodie's abscess of tibia.

Dr. Simmons's Diagnosis

Brodie's abscess of tibia?
Chondroma of tibia?

Anatomical Diagnosis

Localized fibrous osteodystrophy of tibia.

Pathological Discussion

Dr. Mallory: I am sorry Dr. Sumner M. Roberts is not here to describe first-hand his operative findings. He explored this lesion and found a very thin shell of cortical bone. Underneath, and apparently quite separate from the cortical bone, was a spherical tumor mass which was fairly firm and felt rather gritty. This was dissected out and Dr. Benjamin Castleman, who was called over to examine it, thought that it looked in gross very much like the lesions which Jaffe[1] in New York has described as osteoid osteoma (Fig. 1). When the sections came through it

Figure 1. *Tumor and Overlying Cortical Bone.*

was found that the mass consisted of rather acellular fibrous tissue containing numerous narrow trabeculae of bone. In a few areas there was a little active bone formation but for the most part it was completely inactive. We puzzled a great deal over the slides and eventually sent them to Dr. Jaffe who finally made a diagnosis of a localized fibrous osteodystrophy, whatever that may mean. The histologic picture is essentially the same as the one a colleague of Dr. Jaffe's, Lichtenstein,[2] has described in a number of cases presenting the syndrome that Dr. Fuller Albright[3] has made familiar to all of us.

Dr. Hampton: Was there any evidence of inflammation?

Dr. Mallory: Not the slightest. The patient was not worked up as thoroughly as might have been desired, either preoperatively or postoperatively, but so far as is known she had no lesion in other bones and left the hospital perfectly well.

Dr. George W. Holmes: Could this be related to subchondral necrosis? It is pretty close to that region of the tibia.

Dr. Mallory: At the time of examination there was no evidence of necrosis, but that does not rule out the possibility that there may have been some in the past.

DR. HOLMES: Your pathological description sounds very much like the subchondromal necrosis of Pick or osteochondritis.

DR. MALLORY: I feel so relatively unfamiliar with that condition that I do not believe I can rule it out with certainty, although it does not correspond to my impression of osteochondritis.

DR. SIMMONS: The pathologic picture would not fit in with von Recklinghausen's disease or subperiosteal neurofibromatosis?

DR. MALLORY: Again I cannot answer. I see no reason why a primary fibroma occurring within the bone might not show foci of ossification. It is conceivable that that might be the situation here.

DR. HAMPTON: It would not show a dense margin in the bone defect. I cannot imagine a benign lesion in the bone that would produce an area of new bone, 2 cm. in width, around it. Pressure necrosis is not like that. This bone defect has a very definite margin, a thick margin, much thicker than the cysts in Albright's disease. I cannot conceive of a tumor producing this picture. Can you, Dr. Holmes?

DR. HOLMES: No.

DR. MALLORY: Our thought on the whole was that it probably was not neoplasm, but we have not the slightest idea what is back of it. It certainly appears to be a benign lesion, and I have no doubt she is cured.

DR. GRANTLEY TAYLOR: I should like to know about the Hinton test in bone syphilis.

DR. MALLORY: Without having looked the matter up I should be inclined to differ with Dr. Simmons on that, and say that the Hinton test is usually positive in bone syphilis, although I have seen cases in which the Hinton test was negative and the Wassermann positive. There is a slight danger in regard to the Hinton test: it is so sensitive that there is occasionally a pre-zone phenomenon, just as in the Widal reaction, and a negative reading is obtained in a case which really is too strongly positive to show.

DR. SIMMONS: I might say that I was quoting the late Dr. C. Morton Smith when I made the statement.

DR. FULLER ALBRIGHT: Why is this not a Brodie's abscess?

DR. MALLORY: Because it shows no trace of inflammation. I find it hard to believe that an infectious lesion of the bone could heal and not leave behind even a lymphocyte.

DR. ALBRIGHT: I should think it could in time.

DR. HAMPTON: She had clinical evidence of inflammation, and it seems as though the lesion should have been inflammatory.

DR. SIMMONS: Could it not have been a bursa?

DR. MALLORY: Nothing is said in the operative note about a bursa.

DR. SIMMONS: No; but it might have quieted down.

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CASE 25072

PRESENTATION OF CASE

An eighteen-year-old girl was admitted complaining of swelling above the right elbow.

About eight weeks before entry she first noted a lump deep in her right upper arm just above the elbow. It was not tender or painful and had not changed in size. She consulted a physician, following which fifteen doses of x-ray therapy were given over the area. The treatment blanched the skin but did not change the size of the tumor. She had had no other symptoms.

About eleven weeks before admission, during the New England hurricane, she received lacerations of the head and left leg, but remembers no injury or pain involving the right arm. The swelling had interfered in no way with the use of the arm. The past history and family history were noncontributory.

Physical examination showed a well-developed and nourished girl in no distress. The general physical examination was negative except that the skin showed acne over the face, back and chest. The blood pressure was 115 systolic, 60 diastolic. There was a 4 by 5 cm. swelling involving the right humerus just above the elbow. The skin over this area was mottled and depigmented. No axillary nodes could be made out.

The temperature was 98.6°F., the pulse 90, and respirations 20.

Examination of the urine was negative. The blood had a serum-calcium of 11.1 mg. per cent, a phosphorus of 4.2 mg. per cent, and a phosphatase of 3.8 units.

Films brought in by the patient, taken before x-ray treatment, showed an area of destruction with ill-defined margins, involving the lateral cortex of the lower end of the right humerus (Fig. 1). This defect was about 4 cm. in length, and there was no new bone formation or calcification within the soft tissues. The soft-tissue mass was about 5 cm. in diameter. It was not present at the site of bone destruction. The films

taken on admission showed a sharply defined concave defect in the lower end of the right humerus, measuring 4 cm. in length and 1 cm. in depth (Fig. 2). Opposite this defect there was a thin shallow bone which at each end was continuous with the periosteum. This shallow bone

Figure 1. *X-Ray Film before Treatment.*

appeared to be intact in both views. There was elevation of the periosteum for about 1 cm.

The patient's chart remained normal. On the third hospital day an operation was performed.

Differential Diagnosis

Dr. Grantley W. Taylor: An eighteen-year-old girl presented in her arm just above the elbow a mass of eight weeks' duration, which was not tender or painful. She had received a course of fifteen x-ray treatments before she came to the hospital, so that presumably a diagnosis had been made. I do not know, but I infer that the treatment did not affect the lesion. This was essentially a symptomless mass, and the only findings on physical examination that were relevant were the presence of a mass and some skin changes in the vicinity, which I should say were probably attributable to the x-ray treatments she had had. The skin in the area was mottled and depigmented. The laboratory work was essentially noncontributory. The chart was flat and the serum calcium, phosphorus and phosphatase were all within normal limits. The only other relevant preoperative information was contributed by the x-ray films, and I think we might see them before we go farther.

In the original film there is no evidence of repair, regenerative activity or encapsulation and we are dealing simply with an area of destruction chiefly confined to the cortical bone. Dr. Simmons presented in his discussion of the previous case the main pathological categories which we present to ourselves in an attempt to arrive at a diagnosis. The constitutional and systemic diseases, of which hyperparathyroidism is a type, usually give some indication by x-ray of the presence of other bone lesions, or betray their pres-

Figure 2. *X-Ray Film After Treatment.*

ence by abnormal chemical findings in the blood. Reviewing the inflammatory conditions we have osteomyelitis, syphilis, tuberculosis and a few extremely rare diseases. We have nothing in the data at present to justify the assumption that any one of these is present, and we are brought to the fact that we are dealing with a tumor.

The question arises as to whether this is primary or metastatic tumor, and again we have nothing in the record as presented to give us any cause to suppose we are dealing with metastatic malignancy. Of the primary tumors we can be

concerned with a benign or a malignant lesion. To my mind the character of the x-ray film as shown here in no way suggests a benign lesion. The defect is very irregular. Around its margins there is nothing suggesting the lobulations of a chondroma. I think that we are entitled to rule out chondroma or osteoma, giant-cell tumor, and so forth, on the basis of this x-ray appearance, and we are brought to the conclusion that we are dealing with a malignant tumor. I do not believe that we are justified in limiting ourselves wholly to a Ewing's tumor and to osteogenic sarcoma in this differential diagnosis. Parker and Jackson* have recently described reticulum-cell sarcoma as a tumor of bone.

Are there other tumors to which we need to give thought? When I read this description I could not be sure that we were not dealing with a lesion like a single myeloma, which sometimes occurs and presents a confusing picture. However, this lesion is so clearly cortical that I do not believe we can relate it in any way to a disease which is primary in the bone marrow.

The reaction to x-ray treatments is of a good deal of interest. I wonder whether we can say that this ring of bone reaction outside the tumor might have been present originally but that through some technical error in the way the film was taken it failed to show up; I do not picture that type of reaction as an immediate consequence to a series of x-ray treatments. This film shows a rather narrow ring of periosteal bone, with a vague hint of trabeculation on its inner border, and to my mind it raises the question of some sort of bone cyst. I do not know of any sort of bone cyst which would develop in the periosteum in that fashion. I think if we were dealing with a subperiosteal chronic osteomyelitis we should have more suggestion of reactive new bone on the side of the shaft. If we had such a reaction this picture would be consistent with chronic inflammation. The margins of the defect certainly are very ragged in the original film, and in this one quite smooth, which indicates that there has been a distinct effect on the pathologic process as a result of the x-ray and a tendency for bone repair to take place. Now what sort of tumor will respond to fairly extensive x-ray treatment — Dr. Hampton tells me the total dose was about 4000 r. Conspicuously, a Ewing's tumor responds very well to radiation treatment, as does a myeloma or a reticulum-cell sarcoma. However, the x-ray appearance rules out myeloma. I believe that either a reticulum-cell sarcoma or a Ewing's tumor would be perfectly consistent with all the information that we have in this case. I should like to have Dr. Holmes present his views of the x-rays.

*Parker, F., Jr., and Jackson, H., Jr.: Primary reticulum cell sarcoma of bone. Surg., Gynec. & Obst. 68:45-53, 1939.

Dr. George W. Holmes: I know the answer. However, I think it might be interesting if I reviewed my own reaction to this case.

I first heard of this patient through a letter and films from a doctor in Vermont who had the case originally. I had the history about as it has been given and this set of films, with the statement that the patient's parents had refused operation. They would permit a biopsy, but the doctor very wisely refused to do it without permission to operate, so he sent the films to me with the question as to whether she ought to have x-ray treatment. I said at that time, reasoning very much as Dr. Taylor did up to that point, that it could not be a benign tumor and probably was malignant. The patient's other bones were x-rayed and carefully studied for the possibility of a metastatic lesion. I finally came down to the only seemingly possible diagnosis — a primary malignant lesion of bone. I thought treatment was justified so he went ahead and treated her. Then after the treatment was completed they sent her down for a consultation because the family was getting anxious. Dr. Simmons and I saw her, and we could not explain the x-ray picture taken at that time on the basis of x-ray effect. My impression was that the separation of the periosteum from the shaft did not develop during the interval but merely became visible and that the separation of periosteum from shaft was present but not visible at the time of the first examination. I doubt if the deposit of calcium had anything to do with the x-ray treatment; it is the natural course of events with separation of the periosteum.

Dr. Taylor: Do you believe that the tendency of the shaft side to smooth itself was attributable to the same reaction?

Dr. Holmes: I think that the irregularity was due to the same thing that separated the periosteum from the shaft.

Dr. Taylor: In the first film it looks rougher than in the others.

Dr. Holmes: I agree.

Dr. Taylor: What I have been attributing to the result of x-ray treatment is apparently only attributable to the passage of time. What would give rise to the elevation of periosteum — which must have taken place at the time of the original plates — so recently as not to have shown reaction? Is trauma a possible explanation? She was injured in the hurricane, but so far as I can make out the head and the legs suffered whereas the rest of the body did not. Perhaps she had been so confused with the bump on the head that she was

not aware of an injury of the arm. Could a subperiosteal hemorrhage account for the picture? I still do not believe that the shaft should be as irregular as it appears to be from subperiosteal hemorrhage. I am completely at a loss in the matter of making a diagnosis. I still have the impression that perhaps it was a malignant bone tumor, such as a Ewing's tumor, but I say this without any great confidence.

DR. TRACY B. MALLORY: I think that Dr. Taylor has shown wisdom in not committing himself to a definite diagnosis.

DR. CHANNING C. SIMMONS: I might add that this girl was seen by an eminent orthopedic surgeon in this city and one in New York, both of whom made a diagnosis of malignant tumor and advised against operation because they believed it to be incurable. Dr. Ernest A. Codman saw these films and thought it was probably a benign tumor, actual type unknown. He agreed that it might be malignant. I thought it was probably a malignant tumor, type unknown, but did not dismiss the possibility of its being benign. That is as far as anyone got. Since then I have seen a paper describing subperiosteal bone cysts in von Recklinghausen's disease. So far as I could determine she had no evidence of subcutaneous nodules, pigmented areas in the skin or other forms of that disease.

DR. MALLORY: Will you describe your operative findings?

DR. SIMMONS: At operation a subperiosteal cyst containing clear brown fluid was found. The outer wall was composed of fibrous tissue and periosteum with areas of calcification and the inner wall of normal appearing cortical bone. The entire outer wall of the cyst was removed, allowing the muscle to come in contact with the normal appearing bone.

CLINICAL DIAGNOSIS

Sarcoma, right humerus?
Benign bone tumor, right humerus?

DR. TAYLOR'S DIAGNOSIS

Ewing's tumor, right humerus?

ANATOMICAL DIAGNOSIS

Bone cyst, etiology undetermined.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The microscopic examination of the wall of the cyst showed fibrous tissue and a moderate number of foreign-body giant cells, with no evidence of neoplasm, so that the diagnosis remains bone cyst, etiology unknown.

DR. TAYLOR: Is this a possible sequela to trauma with hematoma?

DR. SIMMONS: May I answer that? This girl was injured in the hurricane. A chimney fell through the roof injuring her, killing two other girls and breaking the back of a third one. The doctor who examined her carefully the following day assured me that there was no abrasion on the arm and that she found the tumor at that time.

A PHYSICIAN: Did the x-ray treatment have any effect?

DR. SIMMONS: None at all. Presumably that was done at the psychological moment after the acute reaction had subsided and before the secondary fibrotic changes had taken place.

A PHYSICIAN: Was the wall of the cyst enucleated?

DR. SIMMONS: No; there was no capsule. The outer wall was composed of periosteum with bone in it.

DR. AUBREY O. HAMPTON: The thing I object to is accepting a pathologist's diagnosis in regard to something that is past. How does he know what was here first?

DR. MALLORY: I have not the slightest evidence to offer. I can only guess, as you can.

DR. HAMPTON: It is certainly conceivable that something, perhaps a neoplasm, that had been there was completely destroyed by radiation. The pathologist cannot tell what may have been there before the radiation.

DR. TAYLOR: Do you want to offer a suggestion as to what was there?

DR. HAMPTON: I do not.

DR. MALLORY: I believe this girl is going to remain well.

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CONVALESCENCE

Convalescent care is relatively neglected in the United States. Its importance in the restoration of health does not seem to be properly appreciated by physicians, otherwise there would be greater demand for facilities to care for convalescent patients.

Several pleas have been made in the past that more interest be taken in convalescence, but relatively little progress has been made. A new challenge is at hand — a report of a study of convalescence undertaken by an Advisory Committee on Convalescence of the Boston Council of Social Agencies under the leadership of Miss Ida M. Cannon.[1] In this report it is estimated that 12,000 patients from the wards and outpatient departments of Boston hospitals need convalescent care annually, but in 1937 only 2500 were known to have received it, and a large proportion of these were children for whom facilities are far more adequate than those for adults. There are only three chartered institutions for adult convalescent care in Boston, with a total of 96 beds; of these only 10 are for men and all are for ambulatory patients. In spite of these small facilities none of the institutions are used to full capacity. Although several factors are involved, the chief reason for this seems to be that the profession is not sufficiently interested in convalescence. For economic and emotional reasons a patient is apt to resist a suggestion that two or three weeks be spent at a convalescent home, so that the demand for beds for convalescent patients must be created by the physician. Yet convalescence is not a very interesting subject to the average doctor, and often is given little consideration by those in charge of patients in large hospitals — a group of individuals who particularly need institutional convalescent care.

Undoubtedly fatigue, in one form or another, is a major symptom of a large number of individuals. The role played by so-called mental or physical fatigue, and by altered physiologic states that cause individuals to note they feel fatigued, in the development of disease is not well understood, but undoubtedly fatigue greatly influences the onset and the intensification of many disorders of man. A significant amount of illness could probably be prevented by a proper appreciation of the danger signal — fatigue — and by intensively treating individuals who complain of it. This can often be done successfully in places designed for convalescent patients and especially when the patient's home is unsuitable for the purpose.

The contrast between facilities for convalescent individuals in Great Britain and the United States is indeed striking.[2] In Great Britain there existed, in 1935, 431 homes for convalescent care as against 179 in the United States chartered as such. In Great Britain they are widely distributed, whereas here there are twenty-four states without any facilities, and 50 per cent of the beds are near

New York City. The British homes are used to capacity, 252,000 people receiving care in a year, and during the depression the facilities were uncurtailed, indicating the importance with which they are regarded. In Boston the institutions for convalescent patients connected with three large hospitals have been closed, chiefly for reasons of economy. But would it not be possible that much expense to a general hospital could be saved by transferring many patients to a department for convalescent care before they are well enough to be sent home but after they are past the acute phase of illness? At the present time many recumbent convalescent patients are being cared for in nursing homes — some good, some poor, but all unlicensed and unsupervised.

A committee of the Hospital Council of Boston has just been established to study the recommendations of Miss Cannon's committee. It is to be hoped that a program for the meticulous care of patients during convalescence will be forthcoming.

REFERENCES

1. Cannon, I. M.: *Facilities for Convalescent Care in Boston.* Boston: Research Bureau, Boston Council of Social Agencies, 1938.
2. Gardiner, E. G.: *Convalescent Care in Great Britain.* Social Service Monographs, No. 34. Chicago: University of Chicago Press, 1935.

A COMMENDABLE PLAN

Among the many sociologic and economic programs under way and in contemplation throughout this country those pertaining to medical service are being studied and operated under a great variety of plans. This situation has come about because of an awakened appreciation of the importance of utilizing all useful devices and agencies which may promote the efficiency of scientific medicine.

While differences of opinion respecting some of the plans exist, there is a common belief that every community should have the resources of a hospital available for such cases as may need extradomiciliary treatment. This applies to a large proportion of accidents, to surgical operations and to facilities for diagnosis and certain highly specialized forms of treatment. Medicine today is, in many ways, a science depending on the services of qualified technicians with elaborate chemical and mechanical facilities at hand for the diagnostician, be he either surgeon or internist.

With this understanding, hospitals have been built in comparatively small municipalities and, when well administered, have been blessings for such communities. These hospitals are rarely self-supporting and have been maintained by endowments or contributions of public-spirited citizens. Present financial conditions warrant the fear that the small community voluntary hospital will not be able to meet the requirements of advancing scientific medicine and that the growing proportion of well-educated doctors will not be satisfied with hospital facilities below the standards to which they have been accustomed in their intern days.

It is desirable to keep these small hospitals well above the grade of nursing homes. A step in the right direction is the agreement recently put into effect by the Salem Hospital and the Mary Alley Hospital in Marblehead, whereby the equipment and facilities of the larger institution are made available for the hospital and physician of the smaller community.

This plan seems to be a feasible solution of the problems confronting the small hospitals, for it makes possible a service practically identical with that of an institution prepared to meet almost all the demands imposed on or incident to the practice of scientific medicine. Probably a survey throughout Massachusetts and other states will disclose communities with conditions similar to those in Salem and Marblehead, where the plan outlined might be advantageously adopted.

We congratulate these two communities for their evidence of a broad-minded spirit of collaboration which will, we believe, be a pattern for similar experiments.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

POSTPARTUM HEMORRHAGE

Mrs. M. M., a twenty-nine-year-old primipara at term, entered the hospital early in the morning of February 5, 1925, the membranes having ruptured.

Her family history was noncontributory. The patient gave a history of Vincent's angina, measles and influenza. She had had no operations. Catamenia began at twelve, were regular with a twenty-eight-day cycle and lasted four to five days, with little pain. Her last period was April 28, 1924, making her due for confinement February 1.

She was first seen on June 20, 1924, at which time her heart was rapid but there were no murmurs. Her lungs were clear and resonant; there were no rales. Her blood pressure was 136 systolic, 64 diastolic. A vaginal examination showed the cervix deep in the vagina and soft. The uterus was anterior. Her pregnancy progressed normally until the date of entry.

The patient continued to lose a little amniotic fluid all February 5 and started in indefinite labor about midnight. At 9 a. m. on February 6 the contractions were beginning to come regularly about every three minutes. Examination showed the cervix dilated to admit two fingers and thin, and the head well in the pelvis. At 11 a. m. the cervix was dilated to admit four fingers. At 1 p. m. a forceps was applied to a rotated ODP, after a median episiotomy because of the lack of progress. A male child in excellent condition and weighing 8½ pounds was delivered. The placenta apparently separated immediately but was not extruded. The fundus was not held properly, and a half hour after delivery it was well above the umbilicus. After the placenta, with membranes complete, was delivered intact, by the Credé method, a clot the size of a baby's head followed. Oxytocics were administered, but the uterus did not contract until the pulse had risen to 160 and the blood pressure had dropped to 90 systolic. The patient was transfused with 450 cc. of citrated blood taken from a compatible donor. She made an uneventful convalescence and was discharged from the hospital eighteen days after delivery.

Comment. There is no explanation for the atony that accompanied this delivery except that the fundus was not held properly after delivery. If it had been, it would have been appreciated that the uterus was rising and one would have known that it was filling with blood. Then the placenta might have been delivered before so much blood had been lost. This uterus was not explored because oxytocics and transfusion eventually effected normal contraction. It has been found in many of these cases of hemorrhage that transfusion not only quickly replaces the lost blood but stimulates the uterine muscles to contract.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning February 20:

BRISTOL NORTH

Thursday, February 23, at 4:00 p. m., at the Morton Hospital, Taunton. Subject — Heart Disease: The treatment of "heart attacks" or "cardiovascular emergencies." Instructor: Sylvester McGinn. Lester E. Butler, *Chairman.*

BRISTOL SOUTH (Fall River Section)

Tuesday, February 21, at 4:00 p. m., at the Union Hospital, Fall River. Subject — Medical Complications in Pregnancy. Instructor: James C. Janney. Howard P. Sawyer, *Chairman.*

MIDDLESEX EAST

Tuesday, February 21, at 4:00 p. m., at the Melrose Hospital (Colby Hall), Melrose. Subject — The Indications and Contraindications for Removal of Tonsils and Adenoids. Instructor: Edwin T. Wyman. Walter H. Flanders, *Chairman.*

MIDDLESEX NORTH

Thursday, February 23, at 4:30 p. m., at St. John's Hospital, Lowell. Subject — Syphilis: Latent syphilis — diagnosis and treatment. Instructor: Rudolph Jacoby. William S. Lawler, *Chairman.*

HEART DISEASE VERSUS HEART FAILURE*

The title of this talk, "Heart Disease versus Heart Failure," was chosen in order to point out that heart disease does not necessarily imply heart failure. The term heart disease signifies that some of the working parts of the heart have been damaged, while heart failure signifies that the heart is unable to do its job as it should be done.

The fact that a person has a structural deformity of his heart does not necessarily signify that he is seriously sick or that his life is in danger. Much of the important work

*A "Green Lights to Health" broadcast given by Dr. Herrman L. Blumgart on Wednesday, January 18, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

in the world is accomplished by people with heart disease. To use the example of the automobile, there may be knocks and squeaks in the engine, but used carefully and skillfully, it may provide entirely adequate service to its owner for many years and may actually survive many more smoothly running engines recklessly run and abused. Every physician sees numerous individuals who are heart cripples, not because of a structural deformity of the heart, but because fear and anxiety have paralyzed the patient. Such patients would lead happy and useful lives if they knew how many individuals beyond seventy show evidences of heart disease which have been present but caused no trouble for thirty, forty or even fifty years. As Osler remarked: "The way to live forever is to acquire a chronic disease and take care of it."

Of the infections which damage the heart, the worst enemies are rheumatic fever, syphilis and occasionally certain acute infections such as diphtheria, scarlet fever, tonsillitis, pneumonia and gonorrhea. The natural wear and tear of old age, which in present-day life frequently occurs before it should in the middle years, causes hardening and narrowing of the arteries which supply the heart with blood. This leads to weakness or even failure of the muscular power of this organ. Of the various infectious diseases, rheumatic fever is first in importance. Most frequently it afflicts children, usually after the age of five, and young adults. This disease often comes on with tonsillitis, and there are likely to be migrating pains in the joints and muscles, wrongly called "growing pains," and sometimes redness, tenderness and swelling of various joints, nosebleeds, fever and malnutrition. This infection may cause weakening of the heart muscle and may permanently scar and distort the valves of the heart. In middle adult life we find not only those who have suffered from rheumatic fever, but those who, having had syphilis in earlier years, have felt so well that they have disregarded treatment of the disease, or may not even have known that they had syphilis. During the years of neglect, the infection may silently cause destruction of the root of the large vessel leading from the left pumping chamber and of the heart valve lying very near it. The individual may feel perfectly well for many years until he suddenly becomes aware of symptoms such as loss of breath, pain, palpitation and weakness. The treatment of such patients is discouragingly unsatisfactory, compared to the striking curative value of modern remedies in the early stages of the disease.

Pain may be an important warning sign of disease of the heart. Not infrequently, however, the patient may describe his symptoms as a vague sense of oppression below the breast bone or as a dull, heavy sensation in the shoulders or arms brought on by exertion, emotion, overeating or exposure to cold. One should remember, however, that other organs such as the skin, muscles, ribs and lungs are also situated within the chest and may give rise to similar deceiving symptoms even when there is no real heart disease. Pain in these places may also be due to irritation of nerves or even to diseases of the stomach and gall bladder. On the other hand, symptoms arising in the heart may be felt only in a distant area such as the fingers, the shoulders or the upper part of the abdomen, or may give rise to belching or burning sensations. In any event, such symptoms, particularly when they occur in people of middle age, should make a person consult his physician; for not only may heart disease be suggested by symptoms in other parts of the body, but diseases elsewhere may be discovered because of sensations felt in the region of the heart.

The causes of cardiac pain are numerous, but almost all have to do with interference with the blood supply to the heart muscle. With hardening and narrowing of the arteries of the heart, the amount of blood which can be pumped through these vessels may be enough while a person is resting, though not enough to take care of the increased amount of work the heart is forced to do during exercise or emotion. The appearance of such pain should be regarded by the patient as a red light or danger signal which, if heeded, will prevent an accident, but which, if neglected, may lead to disaster. People who take care not to work too hard or get excited, which may bring on such attacks, and who can lead a quiet, peaceful life, often live for many decades. Sometimes one of the arteries may be completely blocked and give rise to severe, crushing pressure or pain. The heart muscle supplied by such a vessel then degenerates. Even then, after a part of the heart is out of commission, the rest of the heart may be able to take on the job of the injured part, and people have been known to live comfortably for twenty or thirty years after a heart accident of this sort.

Many people with heart disease who try to live more strenuously than is wise for the condition of their hearts have signs of heart failure. The inability of the heart to pump a sufficient amount of blood in a forward direction causes a backing up of blood in the veins and then congestion of the various organs, skin and underlying tissue takes place. Breathlessness, chronic cough with whitish or pinkish sputum, congestion of the liver, giving rise to pain in the upper right portion of the abdomen, swelling of the legs and inability to lie flat in bed are frequently due to heart weakness. By giving the heart sufficient rest, being quiet in bed, and by the skillful use of drugs made possible by the extraordinary advances in scientific medical knowledge, these signs of failure usually disappear. Such people may lead full, happy and successful lives, particularly by giving the heart less work to do, by cutting down bodily activity, by getting enough sleep and rest, by a slowing down of pulse and by loss of weight. Taking digitalis and certain other drugs which remove the accumulations of fluid is an important aid in treatment. Extraordinary advances have also been made in the treatment of heart disease by means of surgery. In spite of the fact that the heart is very sensitive to injury and is situated within the bony cage of the chest, removal of accumulations of fluid surrounding the heart can be accomplished with safety. In certain forms of heart disease the heart becomes surrounded by a tough fibrous sac which interferes with normal pumping. Surgeons are now able to enter the chest and release the heart from this tight, almost bony, envelope. The injection of nerves by alcohol to relieve the pain of what is called angina pectoris and the removal of the thyroid gland to lessen the work of the heart are other surgical procedures by which increased comfort and long life may be provided for certain cases.

* * *

Q. Does the occurrence of a very rapid pulse or palpitation suggest the presence of heart disease?

A. Palpitation, one of the commonest symptoms of heart disease, means a consciousness of the heart beat. Palpitation due to increased rate of contraction may be caused by disease elsewhere, such as too much action of the thyroid gland. This symptom, of course, also comes on in health, with emotion or strenuous exercise, or even on lying down in bed on the left side in the quiet of the night. Some people have occasional extra beats of the heart throughout their life, feeling a "skipping" of the heart. This irregularity is due to the play of nervous impulses, and some people have noticed this from early life to a ripe old age, without its causing any further difficul-

ty. Occasionally, however, and particularly when this irregularity of the heart beat appears for the first time in middle life, it may be a warning sign of some structural abnormality or an increased irritability due, perhaps, to excessive smoking. Another sign of the "nervous heart" may be paroxysms of rapid heart action. The individual, without warning, suddenly experiences an extraordinary pounding of the heart which may last for minutes, hours or even days. Such attacks, which are most disconcerting, are not dangerous and fortunately the physician has effective remedies with which to stop and to prevent them. It is a great tribute to the heart that in spite of such overwork it does not wear out during such an attack or suffer any lasting damage. Nevertheless, people should not disregard such disorders of the heart beat, but rather allow a physician to investigate and explain what they mean.

Q. If a patient has heart disease, what can be done to prevent heart failure?

A. Once heart disease has come on, there is often a considerable period of time before it produces any important interference with the function of the heart. During this period in which there are no symptoms, the problem of the physician and the patient is to prolong this period of perfect well-being and prevent the onset of heart failure. To do this, the physician attempts not only to control the original causes of heart disease but also to treat other conditions which, while not enough in themselves to cause heart failure, may bring about heart failure in an already diseased heart. These conditions include acute infections, overwork, emotional strain, obesity, too little or improper food, anemia, pregnancy and thyroid disease.

Q. Is it bad to be fat if you have heart disease?

A. The longer the belt line, the shorter the life line. High blood pressure is over two and a half times as common among the overweights as among those of average weight, and the death rate from heart disease and cerebral hemorrhage is one and a half times that of those with normal weight and nearly twice that of underweights.

For angina pectoris the difference is even larger. The mortality of overweights from this condition is more than twice that of those with average weights and two and a half times that of underweights. It is clear then that great gains in individual and community health could be brought about if we could cut down the number of fat people.

Q. Has any progress been made in the prevention of heart disease?

A. While great advances have been made in the diagnosis and treatment of heart disease, even more important progress has been made in regard to the prevention of heart disease. Diphtheria, once so prevalent, is now successfully controlled. The causative agent of rheumatic fever is unknown, but it is recognized that overcrowded living conditions, poor nutrition and neglected infections, particularly of the teeth and tonsils, make children more likely to have rheumatic fever. The rising standards of living and more adequate medical care have led to a real lessening in the number of cases of rheumatic heart disease.

The scientific advances in medicine are nowhere better seen than in our knowledge of syphilis. The infecting organism has been discovered, the different ways by which the disease is passed on are recognized, the clinical manifestations of the disease are accurately known, exact diagnostic tests are available, and effective methods of treatment have been worked out. This information, together with the present public-health campaign, should lead to a great reduction in syphilitic heart disease.

Q. Is heart disease increasing?

A. In spite of such progress, an alarming increase in the occurrence of heart disease has been reported by many observers. There are today probably over two million people in this country suffering from some type of heart disease, and statistics seem to show that the death of one of every seven of us will be caused by disability of the heart. Heart disease as the cause of death is more frequent than cancer, pneumonia and tuberculosis combined.

Q. Is this increase of heart disease something to worry about?

A. Careful study of these statistics is reassuring, however, rather than, as is usually thought, a ground for serious worry. Great progress has been made in the prevention and treatment of tuberculosis and the treatment of diabetes and the diseases of infancy and childhood. The effective control of these diseases, improved sanitation and other successful public-health measures have led even during the past twenty-five years to an increased life expectancy of fifteen years. Many people who formerly died of diabetes or died from one of the infectious diseases of childhood attain old age with its tendency toward hardening of the arteries and consequent heart disease. The increase in heart disease is therefore due to the fact that heart disease is mainly a disease of the older years and that over 60 per cent of all deaths now occur after the age of forty-five.

Q. What is coronary thrombosis?

A. Coronary thrombosis is a condition in which one of the arteries which supplies the heart with blood becomes plugged with a firm clot of blood.

Q. Can anything be done to prevent the accident of coronary thrombosis?

A. If people find that they have to cut down on exercise, if they have attacks of pain in their chest and shortness of breath, then they should be kept very quiet so that, if a clot of blood occurs, nature will have a chance to form other blood vessels to take the place of the plugged one. Avoid strenuous physical effort and emotional excitement which, with its attendant increased blood pressure, leads to a greatly increased strain on the heart.

Q. Does physical activity immediately after severe pain affect the amount of damage to the heart muscle?

A. Our latest knowledge indicates that it certainly does. Tying off a blood vessel in dogs causes a larger area of damage in those which are allowed to exercise afterward than it does in those which are kept quiet. Exercising muscle (and the heart is a muscle) requires more oxygen and therefore a larger blood supply than a resting one. This is in keeping with the accumulated experience of doctors.

If every patient suddenly stricken with severe crushing pain were to lie down, there would probably be a decrease in the number of deaths from the disease. If patients followed their doctor's advice and remained at absolute rest in bed for a sufficient time, some deaths and much incapacity would be avoided.

Q. What is the length of time a patient should stay in bed?

A. Healing time is variable — let us say three to eight weeks. Mental and physical rest speeds up the healing process. Eating increases the work of the heart by 50 per cent, and therefore the diet should be light and the patient, if fat, should lose weight.

* * *

Don't worry about heart disease, but if you think you have it, see your doctor and let him tell you what to do, which may very likely be, to stop worrying.

DEATHS

OSGOOD — GEORGE E. OSGOOD, M.D., of St. Petersburg, Florida, died September 21, 1938. He was in his seventy-sixth year.

Dr. Osgood received his degree from Harvard Medical School in 1887. He was a fellow of the American Medical Association and the Massachusetts Medical Society.

WHITE — WILLIAM A. WHITE, M.D., of 249 Warren Street, Roxbury, died February 6. He was in his seventy-fifth year.

Born in Weare, New Hampshire, he received his degree from Tufts College Medical School in 1894. In 1910 he joined the staff of the Massachusetts Women's Hospital, Roxbury, and was on the staff at the time of his death. Dr. White was a former instructor in the history and practice of medicine and lecturer on diseases of children at Tufts College Medical School. He was also professor of materia medica at the Boston Dental College.

His memberships included the Massachusetts Medical Society, the American Medical Association and the Boston Medical Library.

A son, Dr. William A. White, Jr., two daughters, a sister and four grandchildren survive him.

MISCELLANY

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR DECEMBER, 1938

DISEASES	DEC. 1938	DEC. 1937	FIVE-YEAR AVERAGE*
Anterior poliomyelitis	0	1	5
Chickenpox	1096	1670	1482
Diphtheria	23	18	50
Dog bite	561	570	463
Dysentery, bacillary	14	19	4
German measles	55	72	117
Gonorrhea	400	495	556
Lobar pneumonia	415	411	512
Measles	902	349	1148
Meningococcus meningitis	5	4	8
Mumps	470	298	562
Paratyphoid B fever	6	4	1
Scarlet fever	506	926	836
Syphilis	480	475	422
Tuberculosis, pulmonary	177	341	269
Tuberculosis, other forms	23	42	29
Typhoid fever	3	10	11
Undulant fever	5	8	4
Whooping cough	784	732	911

*Based on figures for preceding five years.

RARE DISEASES

Actinomycosis was reported from: Boston, 1; Southbridge, 1; total, 2.

Diphtheria was reported from: Boston, 1; Cambridge, 2; Danvers, 1; Lawrence, 8; Northbridge, 1; Oxford, 1; Plymouth, 1; Salem, 1; Stoneham, 1; Worcester, 6; total, 23.

Dysentery, bacillary, was reported from: Fall River, 5; Northampton, 1; Somerville, 1; Wellesley, 7; total, 14.

Infectious encephalitis was reported from: Boston, 2; Lawrence, 1; Marlboro, 1; Milton, 1; Walpole, 1; Waltham, 1; total, 7.

Malaria was reported from: Foxboro, 2; Springfield, 1; total, 3.

Meningococcus meningitis was reported from: Charlton, 1; Millbury, 1; Newburyport, 1; Rutland, 1; Worcester, 1; total, 5.

Paratyphoid B fever was reported from: Cummington, 1; Framingham, 1; Saugus, 1; Springfield, 3; total, 6.

Pellagra was reported from: Gardner, 1; total, 1.

Septic sore throat was reported from: Amesbury, 3; Boston, 3; Cambridge, 1; Haverhill, 1; Lawrence, 2; Marshfield, 2; Sturbridge, 1; total, 13.

Trachoma was reported from: Boston, 1; total, 1.

Trichinosis was reported from: Boston, 3; total, 3.

Typhoid fever was reported from: Boston, 1; Foxboro, 1; Kingston, 1; total, 3.

Undulant fever was reported from: Boston, 1; Gardner, 1; Lynn, 1; Marlboro, 1; Worcester, 1; total, 5.

For the second time, the first having occurred in December, 1936, not a single case of anterior poliomyelitis was reported. The total incidence for the year 1938 was the lowest ever recorded in Massachusetts.

Chickenpox, measles, German measles, and diphtheria were reported below the five-year average. Except for 1918, scarlet fever showed record low incidence.

Pulmonary tuberculosis was reported at a record low figure. Lobar pneumonia, whooping cough, mumps, tuberculosis (other forms) and meningococcus meningitis were reported below the five-year average. Typhoid fever showed record low incidence. For the second consecutive month, paratyphoid B fever was reported at a record high figure.

Animal rabies showed record low incidence. A previously noted focus in Grafton was still active.

NOTES

At the recent meeting of the History of Science Society held in Chicago, Dr. Henry R. Viets, of Boston, was elected secretary-treasurer.

At a recent meeting of the alumni of Boston University School of Medicine, the principal guest speaker was Dr. Morris Fishbein, editor of the *Journal of the American Medical Association.* Dr. Fishbein stressed the importance of the general practitioner to the medical care of the populace and stated that 85 per cent of sickness is still cared for by the family doctor in his office or in the patients' homes. He complimented the work of the Boston University School of Medicine in developing well-trained general practitioners and in keeping them well informed.

Dr. Hans Mautner, formerly of Vienna, has recently accepted a full-time teaching position as professor of pharmacology in the Middlesex University School of Medicine. He received his M.D. degree from the University of Vienna in 1909, and has devoted twenty-seven years to research at the Pharmacological Institute of the University of Vienna, as a co-worker of Drs. H. H. Meyer and E. P. Pick. Dr. Mautner had been vice-president of the Association of the Vienna Physicians for the past ten years, and had also served as chairman of the Vienna Society of Children's Diseases and was a member of the Board of the Vienna Biological Society and chief of the dispensary for children of the Vienna Herzstation.

The following appointments at Harvard Medical School and Harvard School of Public Health were recently announced: William W. Sargant, of Boston, research fellow in psychiatry, D.P.M. England '36; Charles L. Fox, Jr., of Boston, research fellow in bacteriology, M.D. Long Island '34; Albert M. Moloney, of Boston, assistant in roentgenology, M.D. Tufts '25; James S. Mansfield, of Boston, assistant in medicine, M.D. Harvard '32; Chia-Tung Teng, of Boston, research fellow in medicine, M.D. Peiping Union '33; William M. Hammon, of Brookline, instructor in epidemiology, M.P.H. Harvard '38.

CORRESPONDENCE

TREATMENT OF CHRONIC ALCOHOLISM

To the Editor: I was interested in reading Dr. Bloomberg's article, "The Treatment of Chronic Alcoholism with Amphetamine (Benzedrine) Sulfate," as I had carried on a somewhat similar experiment fifteen or twenty years ago.

I have either mislaid or destroyed my notes, but I suggested buttermilk as a substitute for alcohol in a series of fifty male patients. Only a small percentage reported with any accuracy, but the results in this small group were quite similar to Dr. Bloomberg's findings.

I reached the conclusion that the best results were obtained in those who took my advice most seriously. In other words, psychotherapy rather than buttermilk deserved the credit.

HUGH BARR GRAY, M.D.

Washingtonian Home,
41 Waltham Street,
Boston, Massachusetts.

ALGEBRA AND FRACTURES

To the Editor: Algebra and fractions, yes, but algebra and fractures seem a far cry until we consult the *Century Dictionary* and find the following interesting derivations and definitions:

> Algebra, early modern English, algeber. . . . Mediaeval Latin, algebra, bone setting. . . . Arabic, al-jabr, al-jebr, . . . the redintegration or reunion of broken parts, setting bones. . . . Persian, al-jabr, . . . redintegration, consolidation.

There follows an interesting discussion of the derivation of the component parts of the original Arabic word.

WILLIAM PEARCE COUES, M.D.

12 Monmouth Court,
Brookline, Massachusetts.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of December 16 the following have been accepted:

Abbott Laboratories
- Solution of Epinephrine Hydrochloride 1:1000, 1 fl. oz. bottle
- Solution of Epinephrine Hydrochloride 1:1000, 1 cc. ampule
- Thiamin Chloride-Abbott
  - Tablets Thiamin Chloride-Abbott, 0.33 mg.
  - Tablets Thiamin Chloride-Abbott, 1.0 mg.
  - Tablets Thiamin Chloride-Abbott, 3.3 mg.
  - Ampules Thiamin Chloride-Abbott, 6.66 mg.
- Iodeikon Emulsion Powder-Abbott
- Ampules Estrone, 0.5 mg. in Oil, 1 cc.
- Pentothal Sodium-Abbott
  - Ampules Pentothal 1.0 gm. (15½ gr.), buffered with sodium carbonate 0.6 gm.
  - Ampules Pentothal 0.5 gm. (7½ gr.), buffered with sodium carbonate 0.3 gm.

Lederle Laboratories
- Solution Epinephrine Hydrochloride 1:1000, 1 fl. oz. bottle
- Solution Epinephrine Hydrochloride 1:1000, 1 cc. ampule
- Solution Epinephrine Hydrochloride 1:1000, 5 cc. vial

Eli Lilly & Company
- Tuberculin Ointment (Wolff)-Lilly

The Maltbie Chemical Company
- Ampules Caffeine with Sodium Benzoate, 0.5 gm. (7½ gr.), 2 cc.
- Ampules Sodium Thiosulfate-Maltbie, 10 cc.

The Upjohn Company
- Hypodermic Tablets Strophanthin 0.00033 gm. (1/200 gr.)
- Hypodermic Tablets Digitalin 0.00065 gm. (1/100 gr.)

U. S. Standard Products Company
- Solution Epinephrine Hydrochloride 1:1000, 1 fl. oz. bottle
- Solution Epinephrine Hydrochloride 1:1000, 1 cc. ampule

PAUL NICHOLAS LEECH, *Secretary.*

535 North Dearborn Street,
Chicago, Illinois.

REPORTS OF MEETINGS

BOSTON PATHOLOGICAL SOCIETY

The Boston Pathological Society met at the Evans Memorial Hospital on Thursday evening, November 17, for a symposium on the recent equine encephalitis epidemic. Dr. Charles F. Branch presided.

Dr. Allen Hill reported concerning 8 cases observed at the Children's Hospital, in which the ages of the patients, with the exception of a fourteen-year-old boy, ranged between one and eighteen months. The cases were in three groups: 2 patients who came in violently ill and died within twenty-four hours before a diagnosis could be made — in both cases the diagnosis was established by postmortem examination and the isolation of the virus; 3 patients who also came in critically ill but remained in this condition for five to twenty-one days before dying; and 3 patients who managed to survive the acute illness but were left with severe residual paralysis.

An example of the first group is the case of a fourteen-year-old boy who, after a morning of normal active play, went to lunch with a slight headache. About an hour later he could not be aroused from his afternoon nap, and he was taken to an outlying hospital. Following a period of drowsy arousal he lapsed back into coma. The next day the spinal fluid contained 30 cells per cubic millimeter and gave a positive Pandy test, so he was transferred to the Children's Hospital. On arrival there, thirty-six hours after onset, the child was rigid, with arms flexed tightly across his chest, and responded to nothing. Lumbar puncture showed 2000 cells, 80 per cent of which were polymorphonuclears. There were no red cells or bacteria, and both aerobic and anaerobic cultures were negative. There were no localizing neurologic signs, and the child grew worse, with convulsions and increasing spasticity despite large quantities of paraldehyde and luminal. The temperature rose to 108°F., and the child died twelve hours after admission in a convulsion.

An example of the third group is the case of a twelve-month-old boy who returned from a normal day at the beach and seemed unusually irritable at supper. He played through the evening in his usual manner — despite a

momentary twitching of his left arm three hours after supper — and was put to bed apparently normal. Next morning he was found in coma. He had a brief left-sided convulsion and despite sedation soon developed two more convulsions. The third convulsion lasted two and a half hours and was still in progress when the child arrived at the Children's Hospital; he was moribund and deeply cyanotic, and the right side was rigid, and the left side relaxed. Two cubic centimeters of intravenous luminal was required to stop the convulsions sufficiently to permit normal respiration. The first lumbar puncture, forty-eight hours after onset, showed 246 cells, 45 per cent of which were polymorphonuclears, and the second, six hours later, showed 250 cells with 60 per cent polymorphonuclears. In this child the disease ran a mild and brief course. His temperature, 102°F. on admission, promptly climbed to 105°F., where it remained for forty-eight hours, while the child was in coma and dependent on parenteral fluids. On the third day the temperature began to drop rapidly and by the fourth day was normal, where it remained. At the end of a week he was able to take feedings, though he remained paralyzed on his right side and showed severe inco-ordination of all movements on the left side. After three weeks with only slight improvement, an encephalogram was done which showed a questionable slight enlargement of the ventricles. At present the child still has a right hemiparesis and inco-ordination of movements on the left side.

All the cases were equally abrupt and violent in onset, and all the patients were as critically ill when they arrived at the hospital twenty-four to forty-eight hours after onset. Of the surviving cases, the second patient was in coma for three weeks and remains a pitiable wreck, with generalized spasticity. The third patient is in a cast because of a spastic hemiplegia and is almost completely deaf and apparently blind. One of the patients in the second group ran a temperature between 103 and 106°F. for twenty-one days before he succumbed.

All the cases occurred during August and September and were of a violence comparable only to that of overwhelming sepsis or malignant polioencephalitis. All had two or three convulsions before coming to the hospital, and coma was universal. Vomiting, fever, cyanosis, rigidity and bulging fontanels were almost universal. All had markedly increased intracranial pressure with cell counts ranging from 250 to 2000 cells, 45 to 100 per cent of which were polymorphonuclears. This preponderance of polymorphonuclear cells reversed itself within forty-eight to seventy-two hours so that 60 to 100 per cent were mononuclear cells after that time. Fever ranged between 103 and 107°F. Blood leukocytosis was always present, in some cases as high as 40,000 cells with 85 per cent polymorphonuclears. All the cases surviving beyond the first forty-eight hours developed a peculiar edema, with puffy face and limbs, which did not pit on pressure but was brawny and very slow to subside. (In the 2 instances in which the serum protein was determined, it was found to be well above the edema level.) All developed a considerable pallor due to secondary anemia.

For its violence of symptoms and critical course and for its universal outcome of death or hopelessly discouraging residual paralysis, this disease has been one of the most terrifying we have seen. The 5 patients who died showed pathologic changes consistent with equine encephalitis, and the virus was recovered from 3 cases — in 1 as late as seven days after onset.

Discussion brought out that because of the spinal-fluid findings on admission which necessitated a differential diagnosis of poliomyelitis and septic meningitis, all cases were treated with sulfanilamide until negative spinal-fluid cultures were obtained. Spinal-fluid sugar and chloride determinations were essentially normal. In no case were red-blood cells found in spinal fluid during the early stages.

Dr. LeRoy D. Fothergill reported on the isolation and identification of the virus. While equine encephalitis due to a virus has been known in Europe for a long time, interest in the disease in this country was first aroused by an epidemic among horses in the San Joaquin Valley in California during 1930-1931. Prompt study isolated the virus and proved its etiologic role. The next problem was the mode of transmission. It was first demonstrated that sick and well animals could be kept together without spread of the disease. In 1933 it was shown that the disease could be transmitted from one animal to another by a mosquito — *Aedes aegypti*. Later, eight varieties of the Aedes were found capable of transmitting the disease. Next it was shown that the virus could be recovered from the blood stream of the horse for only a few hours after the onset of the illness and before the temperature rose —thus making the period available for a potentially infective mosquito meal very short. Furthermore it was found that the mosquito could not transmit the disease until four to six days after the blood meal. The reason for this latent period is not known. Once infected the mosquito probably harbors the virus the rest of its life; but it does not transmit the virus to other mosquitoes or its offspring. Later the tick, *Dermacentor andersoni*, was found capable of transmitting the disease, of harboring the virus for a long time and of passing it on to offspring in the eggs. The first epidemic in the East broke out simultaneously in the coastal salt marshes on both sides of Chesapeake Bay. A study of this outbreak demonstrated a virus but one which is immunologically distinct from the virus of the disease in the West. The equine disease caused by the Eastern variety is also characterized by a much more severe course and a mortality rate as high as 90 per cent, in comparison with the 25 per cent mortality of the Western type of disease.

In the present epidemic among human beings, the virus was recovered by injecting a suspension of fresh brain intracerebrally into a mouse. After twenty-four hours the mouse stops eating, its fur becomes rough, and it later develops convulsions which result in death in forty-eight to seventy-two hours. The virus can be recovered from the animal's brain and passed on indefinitely. In this way the virus has been recovered from 8 human cases. As yet it has not been recovered from the blood or spinal fluid. The virus was further identified by protection tests, wherein three animals — a normal mouse, a Western-type immune and an Eastern-type immune — were injected; only the last mouse survived.

Next came the problem of proving the etiology of the disease in those cases which recovered and that of investigating the possibility of sub-clinical infections, of mild unrecognized cases leaving no residual paralysis and of family contacts. The last is unlikely, because people working with the virus for several years have not developed antibodies. Virus-neutralization tests were used for this study. The results so far have shown that the serums from veterinary and family contacts have no neutralizing power, while that from one of the convalescent cases at the Children's Hospital was able to neutralize 100,000 lethal doses.

Discussion brought out that the virus cannot be recovered from the brain of a case in which death is prolonged. Work is being done to discover how early the neutralization test becomes positive. In addition, Dr. Roy F. Feemster of the Massachusetts Department of Public Health is

titrating various parts of the brain to see if the virus has any tendency to localize in special areas.

Dr. John Dingle reported on some of the newer epidemiologic features. While adult man is apparently not susceptible to the disease, the mouse, guinea pig, cat, dog, horse and sheep are. For this reason the disease is ideal for laboratory investigations. Some animals can acquire the disease by subcutaneous injection or even feeding of the virus. Soon this was extended to show that most of the common birds, with the exception of the chicken, are susceptible, among them the pigeon. During the past summer, pigeon breeders all along the Atlantic seaboard noticed an unusual number of deaths for no apparent reason. One of these pigeons was studied, and its brain was shown to contain the Eastern strain of equine encephalitis virus. The pigeon deaths all ceased abruptly with the onset of cold weather and coincidental with the end of the equine epidemic. This raises the question of the role of the pigeon as a reservoir or secondary host and may be the explanation of the simultaneous outbreak on both sides of twenty-five-mile-wide Chesapeake Bay.

Discussion brought out that the present epidemic in Massachusetts and northern Rhode Island involved some 200 horses; that the virus from human cases has been passed on in both pigeons and horses; and that calves get only a mild temperature reaction and no severe illness when inoculated, though an encephalitis of cattle is known in Europe and is due to a different virus. The incubation period after inoculation in the human being is unknown but judging from animal experiments is probably between two and five days. The major missing link in the tale, so far, is failure to find mosquitoes or ticks which harbor the virus. But the circumstantial evidence of no contact cases, seasonal incidence and cases separated by several miles is strong, and the statistical probability of finding such insects in the field very slight. Where the disease is harbored during the winter, the natural reservoir and possibility of other intermediate hosts and the possibility of carriers constitute the important unsolved problems.

Dr. Sidney Farber presented the pathologic findings and pointed out that much of his work was excellently supplemented and confirmed by that of Dr. Charles F. Branch. Grossly the brains showed nothing distinctive aside from considerable edema, with an increase in weight, and an intense congestion most striking in the pons, medulla and basal ganglia. All 5 cases showed a pressure cone.

Microscopically there were four outstanding features. (1) Nerve cells throughout the brain showed Nissl-substance breakdown, chromatolysis and general cellular dissolution, particularly in the pons, medulla and basal ganglia. Many of the nerve cells were surrounded by polymorphonuclear cells, and some were undergoing phagocytosis. There was also some increase in the number of glial cells. Despite the large numbers of leukocytes no bacteria could be demonstrated by culture or special stains. (2) There were large accumulations of inflammatory cells in the perivascular spaces of Virchow-Robin. (3) There was a diffuse meningeal infiltration with inflammatory cells most noticeable about the base of the brain. In 1 case, the changes were striking enough to lead to a diagnosis of basilar meningitis when the brain was first removed. The inflammatory cells in these neuronophagic, perivascular and meningeal infiltrations paralleled the spinal-fluid findings at the time of death. During the early stages when most of the cells in the spinal fluid were polymorphonuclear leukocytes, the inflammatory reaction proved to consist chiefly of these cells, and later, when the spinal fluid showed chiefly mononuclear cells, the infiltration was chiefly mononuclear. (4) A striking and widespread arteritis was present with infiltration of the vascular walls by polymorphonuclear leukocytes and a fibrin network. Yet hemorrhages were quite uncommon though a few tiny ones could usually be found.

These lesions were found widespread and most numerous throughout the pons, medulla and basal ganglia. The cortex showed a diffuse but patchy involvement with no normal areas adjoining the involved areas. Usually the cerebellum was completely spared. The spinal cord showed edema and congestion and a moderate degree of nerve damage but no perivascular or meningeal infiltration and no inflammatory exudate. Hence there was no myelitis in the strict sense of the word. However, this cord sparing may be accidental in a limited number of cases, and further study may show a true encephalomyelitis.

In the lungs, both gross and microscopic edema were invariably present. There was also a definite infiltration of the alveolar walls with polymorphonuclear and mononuclear cells, but no bacteria could be demonstrated. This indicates a definite early pneumonitis such as is frequently seen in virus diseases and raises a serious question as to the exclusive neurotropism of the virus. No inclusion bodies were found, but we no longer require them to diagnose a virus disease. The kidneys showed congestion and petechiae and, together with the heart and pancreas, terminal thrombi. In 1 case the thrombosis of a large sinus was associated with a subarachnoid hemorrhage.

These microscopic lesions differ from those of the St. Louis and Japanese Type B encephalitis by virtue of the massive early polymorphonuclear infiltration and the arteritis. Poliomyelitis does not show the massive brain involvement and distribution present here; nor does the disease under discussion show the anterior-horn-cell lesions that are characteristic of poliomyelitis. Encephalitis lethargica, a more chronic disease, shows chiefly a lymphocytic infiltration, with little diffuse brain involvement and meningeal reaction. There is none of the demyelination in this epidemic such as one sees in Schilder's disease and postpertussis and postmeasles encephalitides. This epidemic is the most acute and violent encephalitis we have ever seen. In one of Dr. Branch's cases there was massive destruction of a large amount of nervous tissue, severe cord lesions and widespread thrombosis of many vessels.

Dr. Leo Alexander mentioned 4 cases seen at the Boston City Hospital. Postmortem examination was delayed too long to recover the virus in any case. He emphasized the high total protein with normal chlorides and sugar in the spinal fluid and the marked involvement of the mammillary bodies, paraventricular nuclei and the region about the third ventricle.

EASTERN HAMPDEN MEDICAL ASSOCIATION

The fifty-ninth annual meeting of the Eastern Hampden Medical Association was held on Thursday, February 2, at the Oaks Hotel in Springfield.

The following officers were elected for 1939: president, Dr. Michael J. Kranichuck, of South Hadley Falls; vice-president, Dr. J. Joseph Klar, of Springfield; secretary-treasurer, Dr. James J. Grace, of Springfield.

The retiring president, Dr. T. H. McSweeney, delivered the annual discourse "Family Doctor or Federal Agent?"

The treasurer's report showed the society to be in better financial condition than for several years past.

Following the annual dinner served to the members, their wives and guests, an elaborate entertainment was presented in the auditorium.

J. Joseph Klar, M.D., *Secretary-Treasurer.*

NOTICES

REMOVAL

HENRY M. BAKER, M.D., announces the removal of his office to 353 Commonwealth Avenue, Boston.

HOSPITAL RESEARCH COUNCIL

The Hospital Research Council will hold a meeting in the Ether Dome of the Massachusetts General Hospital on Tuesday, February 28, at 5:00 p. m. The program will be as follows:

STUDIES PERTAINING TO THE PHYSIOLOGY OF NORMAL JOINTS

The Origin and Nature of Normal Synovial Fluid. Dr. Marian W. Ropes.
The Removal of Proteins and Aqueous Solutions from Normal Joints. Dr. Walter Bauer.
The Passage of Proteins and Pneumococci from the Vascular System into Joints. Dr. Granville A. Bennett.
The Relation of These Studies to the Metabolism of Cartilage. Dr. Eric G. L. Bywaters.

HENRY K. BEECHER, M.D., *Secretary.*

SALEM HOSPITAL CONFERENCES

There will be conferences at the Salem Hospital every Friday morning at 9:00 until further notice. Physicians are cordially invited to attend.

The form of the conferences for each month will be as follows:

First	Friday	— Grand ward rounds.
Second	"	— Clinicopathological conference.
Third	"	— Grand ward rounds.
Fourth	"	— Clinical conference.

If there is a fifth Friday in any month, there will be either grand ward rounds or a tumor-clinic teaching conference.

SOUTH END MEDICAL CLUB

The next meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, February 21, at 12 o'clock noon.

Dr. Howard F. Root will speak on "Hypertension and Its Complications."

Physicians are cordially invited to attend.

JOHN B. HALL, M.D., *Secretary.*

CARNEY HOSPITAL

The monthly clinical meeting and luncheon of the Carney Hospital will be held in the Andrew Carney Assembly Room on Monday, February 20, at 11:30 a. m.

PROGRAM

Case reports.
Infections of the Foot: Usual anatomical locations and treatment. Dr. John G. Arent. Discussion by Drs. Archibald McK. Fraser, John L. Doherty and John J. Todd.

Physicians and medical students are cordially invited to attend.

ROY J. HEFFERNAN, M.D., *Secretary.*

MASSACHUSETTS ITALIAN MEDICAL SOCIETY

The regular monthly meeting of the Massachusetts Italian Medical Society will be held at the Hotel Kenmore, Boston, on Friday evening, February 24, at 9:00.

PROGRAM

Pathology of Peptic Ulcer. Dr. Marino F. Vidoli.
Sociomedical Problems in Italy. Marquis Carlo De Constantin de Chateauneuf, Italian Consul General of New England.

A general discussion will follow. The medical and allied professions are cordially invited to attend.

CARL F. MARALDI, M.D., *Secretary.*

CAMBRIDGE HOSPITAL

The regular clinicopathological meeting of the staff of the Cambridge Hospital will be held at the hospital, 330 Mt. Auburn Street, Cambridge, on Tuesday, February 21, at 8:30 p. m.

Dr. Elliott C. Cutler will speak on "Surgical Treatment of Peptic Ulcer."

All members of the medical profession are cordially invited to attend.

STEPHEN M. BIDDLE, M.D., *Secretary.*

NORFOLK DISTRICT MEDICAL SOCIETY

The regular meeting of the Norfolk District Medical Society will be held in the Hotel Somerset, Boston, Tuesday evening, February 28, at 8:30. Tel. KEN. 2700.

PROGRAM

The Huntington Hospital and the Cancer Problem. Dr. Joseph C. Aub.
Kodachrome Views of Contagious Disease. Dr. Edwin H. Place.
Collation.

FRANK S. CRUICKSHANK, M.D., *Secretary.*

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, February 23, in the amphitheater of the Peter Bent Brigham Hospital, Dr. Marshall N. Fulton, associate in medicine, Harvard Medical School, and physician, Peter Bent Brigham Hospital, will give a medical clinic. Practitioners and medical students are cordially invited to attend.

BOSTON MEDICAL HISTORY CLUB

The Boston Medical History Club will meet at the Boston Medical Library, 8 Fenway, Boston, on Monday evening, February 20, at 8:15.

Dr. Leroy M. S. Miner will talk on "The Development of Our Knowledge of the Diseases of the Teeth."

Members of the medical profession and other interested persons are cordially invited to attend.

PAUL D. WHITE, M.D., *President,*
BENJAMIN SPECTOR, M.D., *Secretary.*

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, February 20

Monday, February 20

*11:30 a. m. Carney Hospital. Monthly clinical meeting and luncheon.

*8:15 p. m. Boston Medical History Club. Boston Medical Library, 8 Fenway.

Tuesday, February 21

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. Allergy Clinic with Case Presentation. Dr. E. A. Brown.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

*12 m. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

*8:30 p. m. Cambridge Hospital. Clinicopathological meeting of staff.

Thursday, February 23

8:30-9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and Children's hospitals, held this week at the Children's Hospital Orthopedic.

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. Medical-Social-Service case presentation. District Service and Social-Service staffs.

*3:30 p. m. Medical clinic at the Peter Bent Brigham Hospital.

Friday, February 24

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. The Present Status of Specific Therapy for Pneumonia. Dr. Maxwell Finland.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

*9 p. m. Massachusetts Italian Medical Society. Hotel Kenmore, Boston.

Saturday, February 25

*9-10 a. m. Joseph H. Pratt Diagnostic Hospital. Hospital case presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff rounds of the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

Sunday, February 26

4 p. m. Illustrated, public, health lecture, Faulkner Hospital, auditorium. Tuberculosis in This Community. Dr. David Halbersleben.

4 p. m. Free public lecture, Harvard Medical School, Amphitheater of Building D. Nervous Breakdowns. Dr. Vernon P. Williams.

*Open to the medical profession.

February 17 — Urological Conference. Massachusetts General Hospital.

February 19 — Lecture at the Faulkner Hospital. Page 971, issue of December 15.

February 19 — Free Public Lecture, Harvard Medical School. Page 1056, issue of December 29.

February 19 — Beverly Hospital Public Health Lecture. Page 1056, issue of December 29.

February 19 — Salem Hospital Public Health Lecture. Page 126, issue of January 19.

February 20 — Carney Hospital monthly clinical meeting and luncheon. Page 312.

February 20 — Boston Medical History Club. Page 312.

February 21 — South End Medical Club. Page 312.

February 21 — Cambridge Hospital. Clinicopathological meeting of the staff. Page 312.

February 22 — Alumni Day, New York University College of Medicine. Page 173, issue of January 26.

February 23 — Medical clinic at the Peter Bent Brigham Hospital. Page 312.

February 24 — Massachusetts Italian Medical Society. Page 312.

February 27 — New England Heart Association. Page 267, issue of February 9.

February 28 — Hospital Research Council. Page 312.

March 9 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

March 9-11 — New England Hospital Association. Page 267, issue of February 9.

March 13 — Fourth Annual Postgraduate Institute. Page 938, issue of December 8.

March 15, May 15, August 5 and October 6 — American Board of Ophthalmology. Page 126, issue of January 19.

March 27-31 — American College of Physicians. Page 36, issue of July 7.

May 7-15 — International Congress of Military Medicine and Pharmacy. Page 501, issue of September 29.

May 15-16 — American Board of Obstetrics and Gynecology, Inc. Page 218, issue of February 2.

May 15-19 — American Medical Association. St. Louis, Missouri.

June 6, 7, 8 — Massachusetts Medical Society. Worcester.

June 12-17 — Symposium on the Public-Health Significance of the Virus and Rickettsial Diseases. Page 125, issue of January 19.

June 26-29 — National Tuberculosis Association. Page 936, issue of December 8.

September — Boston Psychoanalytic Institute. Page 450, issue of September 22.

September 11-15 — American Congress on Obstetrics and Gynecology. Page 938, issue of December 8.

September 15-28 — Pan-Pacific Surgical Association. Page 863, issue of November 24.

Fall, 1939 — Temperature Symposium. Page 218, issue of February 2.

District Medical Societies

ESSEX SOUTH

March 1 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John Rock. Subject: Endocrinology.

April 5 — Addison Gilbert Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Ethan Allan Brown. Subject: Allergy.

May 10 — Annual meeting. Salem Country Club, Peabody.

NORFOLK DISTRICT

February 28 — Page 312.

SUFFOLK

March 29 — Joint meeting with New England Pediatric Society, Boston Medical Library, 8:15 p. m. Program and speakers to be announced.

April 26 — Annual meeting in conjunction with Boston Medical Library, at 8:15 p. m. Election of officers. Program and speakers to be announced.

WORCESTER

March 8 — Worcester Memorial Hospital.

April 12 — Worcester Hahnemann Hospital.

May 10 — Worcester Country Club — Annual meeting.

With the exception of the annual meeting in May, all the meetings begin with a supper at 6:30 p. m., which is followed at 7:30 p. m. by the business and scientific sessions.

BOOKS RECEIVED FOR REVIEW

Schafer's Essentials of Histology: Descriptive and practical for the use of students. H. M. Carleton. 618 pp. Philadelphia: Lea & Febiger, 1938. $5.00.

Infections of the Hand: A guide to the surgical treatment of acute and chronic suppurative processes in the fingers, hand and forearm. Allen B. Kanavel. Seventh edition. 503 pp. Philadelphia: Lea & Febiger, 1939. $6.00.

Sir Thomas Roddick: His work in medicine and public life. H. E. MacDermot. 160 pp. Toronto: The Macmillan Co. of Canada, Ltd., 1938. $2.00.

Principles of Hematology. Russell L. Haden. 348 pp. Philadelphia: Lea & Febiger, 1939. $4.50.

The Language of the Dream. Emil A. Gutheil. 286 pp. New York: The Macmillan Co., 1939. $3.50.

A Textbook of Neuro-Radiology. Cecil P. G. Wakeley and Alexander Orley. 336 pp. Baltimore: William Wood & Co., 1938. $8.00.

Alcohol in Moderation and Excess: A study of the effects of the use of alcohol on the human system. J. A. Waddell and H. B. Haag. 184 pp. Richmond: The William Byrd Press, Inc., 1938. $1.00.

Emotions and Bodily Changes: A survey of literature on psychosomatic interrelationships, 1910-1933. H. Flanders Dunbar. Second edition. 601 pp. New York: Columbia University Press, 1938. $5.00.

Out of the Running. G. Gertrude Hoopes. 158 pp. Springfield, Illinois, and Baltimore: Charles C Thomas, 1939. $2.00.

St. Thomas's Hospital Reports. Edited by O. L. V. S. De Wesselow and C. Max Page, assisted by N. R. Barrett, J. St. C. Elkington and A. J. Wrigley. Vol. 3, ser. 2. 240 pp. London: St. Thomas's Hospital, 1938.

Immunity: Principles and application in medicine and public health. Hans Zinsser, John F. Enders and LeRoy D. Fothergill. Fifth edition of *Resistance to Infectious Diseases.* 801 pp. New York: The Macmillan Co., 1939. $6.50.

Midwifery. By ten teachers, under the direction of Clifford White. Edited by Comyns Berkeley, Clifford White and Frank Cook. Sixth edition. 676 pp. Baltimore: William Wood & Co., 1938. $6.00.

Petite Chirurgie et Technique Médicale Courante. G. Roux. 591 pp. Paris: Masson et Cie, 1938. 90 Fr. fr.

La Ponction Sternale: Procédé de diagnostic cytologique. P. Émile-Weil and Suzanne Perlès. 183 pp. Paris: Masson et Cie, 1938. 75 Fr. fr.

Roentgen Diagnosis of the Extremities and Spine. Albert B. Ferguson. 435 pp. New York: Paul B. Hoeber, Inc., 1939. $12.00.

A Medical Survey of the Republic of Guatemala. George C. Shattuck. 253 pp. Washington, D. C.: Carnegie Institution of Washington, 1938. $2.50, paper; $3.00, cloth.

A Manual of Fractures and Dislocations. Barbara B. Stimson. 214 pp. Philadelphia: Lea & Febiger, 1939. $2.75.

The Patient Is the Unit of Practice. Duane W. Propst. 219 pp. Springfield, Illinois, and Baltimore: Charles C Thomas, 1939. $3.50.

Dunant: The story of the Red Cross. Martin Gumpert. 323 pp. New York: Oxford University Press, 1938. $2.50.

Body Menders. James Harpole. 296 pp. New York: Frederick A. Stokes Co., 1939. $2.75.

Man and His Body. Howard W. Haggard. 594 pp. New York and London: Harper & Brothers, Publishers, 1938. $4.00.

Surgical Pathology of the Diseases of the Mouth and Jaws. Arthur E. Hertzler. 248 pp. Philadelphia, Montreal and London: J. B. Lippincott Co., 1938. $5.00.

BOOK REVIEWS

Our Common Ailment. Constipation: Its cause and cure. Harold Aaron. 192 pp. New York: Dodge Publishing Co., 1938. $1.50.

It is probably true that informed persons agree with the writer of this book that 10 per cent of the people are more or less constipated and that in a large proportion of these cases the ailment is a functional rather than an organic disease, which may be corrected under intelligent and persistent management. It is, however, too often found that people who should strive for normal behavior of the bowels are impatient and seek relief by taking drugs which tend to establish chronicity. However that may be, Dr. Aaron believes that a proper understanding of the causes and treatment of this trouble will be appreciated by those who really want to secure a return to normal conditions. With this in view he expects that many people will be led to profit by his advice.

In order to enable the patient to prevent and cure bad bowel habits the author first gives a description of the anatomy and physiology of the digestive organs. He then discusses the factors that must be considered in order to differentiate organic and functional disease; for if the former is confused with the latter dire results are liable to follow. Emphasis on prevention is stressed. This depends in large measure on the recognition of parental responsibility with respect to the education of the child. Adequate space is given to the futility and danger of self-medication, and the approved methods to be employed in bringing about restoration of normal habits are set forth with warnings against harmful drugs and the employment of quacks. Instructive chapters deal with hemorrhoids and colitis.

The book is well written, and there is little opportunity for criticism, for most of the opinions of the author will be endorsed by well-informed physicians. Interspersed in the text the reader will find humor and sarcasm, which tend to emphasize the arguments advanced. Although the book is written especially for the non-medical person there is enough of science to interest those physicians who have not given much attention to this subject. Doctors may recommend this book to their patients with propriety. The only objection to it will come from quacks and manufacturers of proprietary drugs.

The Physiology of Anesthesia. Henry K. Beecher. 388 pp. London, New York and Toronto: Oxford University Press, 1938. $3.75.

Dr. Beecher approaches the subject as an expert clinical anesthetist, as a teacher of anesthesia and especially as one who has made a thorough study of the physiological basis of the problem in all its varied aspects.

The book begins with a study of the primary effects of anesthesia, namely the effects on the nervous system as a whole. Herein are considered the meaning of anesthesia and narcosis and the various theories of anesthetic action. Next is the section on respiration, in which the chemistry of gaseous exchange, the behavior of the respiratory center, the viscosity of air and the related problems of obstruction of the airway are considered. Next is the section on circulation, in which the effects of various anesthetics on the heart and on other elements in the vascular system are treated. The final chapter deals with "Organic Effects of Anesthetic Agents." This includes a discussion of blood changes — those in the cells, physical properties and blood chemistry — and a consideration of the effects of anesthetics on the liver and other vital organs and finally on metabolic rate and temperature.

This scholarly work should serve a variety of useful purposes. It should be of great value to the medical student learning the foundations of this important subject. It should serve the professional anesthetist, providing timely warnings of the hazards involved in the use of certain anesthetics or in the use of any anesthetics without critical appraisal of danger signs. It should serve the surgeon in like manner, for he, as well as the anesthetist, should understand fully what is involved in the problem which he shares with the anesthetist. If it were not already obvious, this book should furnish convincing evidence of the importance of teamwork between surgeon and anesthetist. The book is also full of significant material for the physiologist. In the discussion of the relation between the chemical properties of the anesthetics and their action on nervous tissue, and in the related consideration of the theories of anesthesia, there are many important clues which may ultimately lead to a better understanding of the nervous system.

Spinal Anesthesia. Louis H. Maxson. 409 pp. Philadelphia, London, New York and Montreal: J. B. Lippincott Co., 1938. $6.50.

This is a very valuable monograph on a most important aid to safe and successful surgery. The writer discusses in detail the development of spinal anesthesia from its earliest days, in its physiological, pharmacological and technical aspects. His own methods of choice are described extensively, with the reasons therefor. Constant comparisons of the ideas and methods of others of authority in this field are made. An excellent bibliography is appended.